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REPORT OF A SURVEY OF CHILDREN BORN IN 1941 WITH REFERENCE TO CONGENITAL ABNORMALITIES ARISING FROM MATERNAL RUBELLA.

By P. R. PATRICK,

Chief Medical Officer, School Health Services, Department of Health and Home Affairs, Queensland.

DURING the months of June to September, 1947, the Queensland School Health Services conducted a survey of children born in 1941, with a view to determining the extent of abnormalities resulting from maternal rubella during pregnancy. The survey was carried out at the request of the Honourable T. A. Foley, M.L.A., former Minister for Health and Home Affairs, following reports that the large increase in deaf children admitted to the School for the Blind and Deaf, Brisbane, resulted from the epidemic of rubella in 1940. Preliminary work was commenced by the late Dr. L. St. Vincent Welch, at that time Chief Medical Officer, School Health Services, Queensland.

The nursing staff of the School Health Services obtained the names of children required from the school registers and distributed copies of the following *questionnaire* to the mothers of these children:

> Public Health Authorities are alarmed at the number of children born in 1941 who show signs of deaf mutism, heart disease, cataract, or even mental defectiveness.
>
> It is noted that a severe outbreak of German measles occurred in Queensland in 1940, and the medical officers of the Department of Health have been requested by the Honourable the Minister to make a survey respecting all children born in 1941.
>
> From the school register, it is learned that you are the mother of a child born in that year.
>
> Will you therefore be so kind as to afford the Department your most valued co-operation by filling in the replies to the questions listed below, and at your earliest convenience returning the paper in care of your child. Such action may assist in preventing the tragedy of further cases of physical defects in future generations of infants.
>
> i. Did you suffer from German measles in 1940?
> ii. If you did so suffer, were you pregnant at the date of the attack of German measles?
> iii. If you were pregnant, how many months were you pregnant at the date of the attack?
> iv. Has your child shown any physical defects, especially those mentioned in the beginning of my letter?
>
> The symptoms of German measles are:
>
> Pale pink rash clustered in small groups—first on face and neck extending to trunk and limbs. On second day, the face is completely covered by rash. Sometimes there is no fever at all. There is a tender swelling of the glands behind the ear and back of neck; they may reach the size of small peas. The glands in the armpit and groin frequently enlarge.

In all, 9674 *questionnaires* were delivered, and of these, 7822 were returned completed.

From the Registrar-General of Births for Queensland it was learned that 21,509 children were born in Queensland in 1941.

It is seen that information was received concerning at least one-third of these children—enough to allow some idea to be formed of the extent of the epidemic and its resultant effects.

Schools attended by these children included State primary schools, denominational primary schools, opportunity schools and grades, the School for the Blind and Deaf, private schools and institutions.

Altogether there were 262 cases in which mothers were certain they had contracted rubella during that particular pregnancy. Of these, 134 were in the Brisbane area; the remainder of the children attended country schools throughout Queensland. Of those in Brisbane schools, 129 were examined clinically, the remainder being unavailable for examination.

Clinical Findings.

The clinical notes of these 129 children are presented below in Table I.

The division into grades of deafness is that published in the "Report of the English Committee of Inquiry into Problems Relating to Children with Defective Hearing".[1] It is as follows:

Grade I: Children with defective hearing who can, nevertheless, without special arrangements of any kind, obtain proper benefit from the education provided in an ordinary school, elementary, secondary or technical.

Grade II: Children whose hearing is defective to such a degree that they require for their education special arrangements or facilities, but not the educational methods used for deaf children without naturally acquired speech or language. These facilities range from a favourable position in the ordinary school classroom to attendance at a special class or school.

Grade IIA: Those children within Grade II who can make satisfactory progress in ordinary classes in ordinary schools provided they are given some help, whether by way of favourable position in class, by individual hearing aids, or by tuition in lip-reading.

Grade IIB: Those children within Grade II who, even with the help of favourable position in the class, individual hearing aids or tuition in lip-reading, fail to make satisfactory progress in ordinary classes in ordinary schools.

Grade III: Children whose hearing is so defective and whose speech and language are so little developed that they require education by methods used for deaf children without naturally acquired speech or language. This grade includes the totally deaf.

Of the 129 children examined, 51 had some abnormality. Of these, at least 37 could be classified as having a serious defect. These include all deaf mutes (Grade III deafness), mental deficients, congenital cardiac patients and children with cataracts. Eleven children had two or more serious abnormalities. These combinations were: (*a*) deaf-mutism *plus* congenital heart disease; (*b*) mental deficiency, congenital heart disease and cataract. In addition, less serious defects were found to be present in the deaf-mutes examined.

Table II shows the number of the various defects found.

Abnormalities in Other Children.

An examination was made of 1000 children attending State schools whose mothers were certain that they had not suffered from rubella during that pregnancy. In addition all other deaf children born in 1941 attending the School for the Deaf and Blind were examined. In the first group the abnormalities stated in Table III were found.

Of 40 children born in 1941 attending the School for the Deaf and Blind, 26 gave a history of maternal rubella. Several of these had other abnormalities. The remaining 14 had no other abnormalities. Of these 14, in 11 cases the parents stated that their child was born deaf. Meningitis, hereditary factors and morbilli were given as the causes in the remaining three.

Country.

From the country there were 3766 replies to 4415 *questionnaires* delivered. Amongst those replies, there were 128 cases in which mothers stated that they had suffered from rubella in pregnancy. Of these 128 cases, 36 mothers stated that their children had congenital abnormalities. It was impossible to examine these children owing to the great distances between the patients. An accurate estimation of the degree of the abnormality cannot be given; but in this respect it may be pointed out that in the Brisbane area, in which nearly all the patients were examined, there was no great discrepancy between the mothers' statements and the clinical findings when the children were examined.

As in the city, the preponderant abnormality was again deafness. Of 36 children stated to have abnormalities, 19 were said to suffer from varying degrees of deafness. There were eight cases of eye abnormality, and three

Table I.

Case Number.	Period of Pregnancy at which Rubella was Contracted.	Clinical Notes and Remarks.[1]
1	About conception.	Grade III deafness. Family history of mental backwardness, but this child seemed mentally bright.
2	At conception.	Grade III deafness. Systolic murmur at apex. Attending School for Blind and Deaf.
3	At conception.	No congenital abnormality detected.
4	Three weeks.	No congenital abnormality detected.
5	One month.	Backward at school, but not definitely subnormal. No other congenital abnormality detected.
6 and 7	One month.	No congenital abnormality detected.
8 } Twins	Two weeks.	First child, no congenital abnormality detected.
9 }		Second child not examined, but mother states child in mental home; was uncontrollable at home.
10	One month.	No congenital abnormality detected.
11		
12		
13		
14	Two months.	No congenital abnormality detected.
15		
16		
17		
18		
19	Two months.	Grade IIB deafness. Otherwise no congenital abnormality detected. Attending State school.
20	Two months.	Has high frequency deafness, but otherwise no congenital abnormality detected. Progressing normally at State school.
21	Six weeks.	No congenital abnormality detected.
22	Two months.	Grade IIA deafness. Otherwise no congenital abnormality detected. Attending State school.
23	Two months.	Grade III deafness. Pulmonary systolic murmur; very poor physique. Attending school for blind and deaf.
24 }	Two months.	Grade III deafness. Otherwise no congenital abnormality detected. Attending
25 }		School for Blind and Deaf.
26	One to two months.	No congenital abnormality detected.
27		
28		
29		
30		
31		
32	Three months.	No congenital abnormality detected.
33		
34		
35		
36		
37		
38	Three months.	High-frequency deafness. Progressing normally at State school.
39	Three months.	Malformed right ear lobe, but otherwise no congenital abnormality detected.
40	Two to two and a half months.	Grade III deafness. Appears mentally deficient as well. Attending opportunity grade.
41 } Twins	Two to three	No congenital abnormality detected.
42 }	months.	
43	Three months.	Mentally deficient. Hearing, vision, heart normal. Teachers say unable to be taught.
44	Two and a half months.	No congenital abnormality detected.
45		
46		
47		
48		All Grade III deafness. Otherwise no con-
49	Three months.	genital abnormality detected. Attending
50		School for Blind and Deaf.
51		
52		
53		
54	Three months.	Grade III deafness. Pulmonary systolic murmur and pulmonary second sound accentuated. Attending School for Blind and Deaf.
55	Two and a half months.	Grade III deafness. Attending School for Blind and Deaf.
56	Two months.	Grade III deafness. Attending School for Blind and Deaf.
57	Three and a half to four months.	No congenital abnormality detected.
58		
59		
60	Four months.	No congenital abnormality detected.
61		
62		
63	Four months.	Mentally deficient. Unable to be taught. Vision, hearing, heart normal.
64	Three and a half to four months.	No congenital abnormality detected.
65	Four months.	Grade I deafness. Otherwise no congenital abnormality detected.
66	Four months.	Grade I deafness, left ear. Otherwise no congenital abnormality detected.

[1] *Vide infra*, explanation of grades of deafness.

Table I.—*Continued.*

Case Number.	Period of Pregnancy at which Rubella was Contracted.	Clinical Notes and Remarks.[1]
67	Three to four months.	Grade I deafness. Otherwise no congenital abnormality detected.
68	Four months.	Mitral systolic murmur. Otherwise no congenital abnormality detected.
69	Two and a half months.	Grade III deafness. Otherwise no congenital abnormality detected. Attending School for Blind and Deaf.
70	Three months.	Grade III deafness. Pulmonary systolic murmur. Attending School for Blind and Deaf.
71	Three to four months.	Grade III deafness. Pulmonary systolic murmur. Attending School for Blind and Deaf.
72	Three to four months.	Grade III deafness. Otherwise no congenital abnormality detected. Attending School for Blind and Deaf.
73	Three months.	Grade I deafness. Otherwise no congenital abnormality detected. Progressing normally at State school.
74, 75	Three and a half months.	No congenital abnormality detected.
76	Three and a half months.	Astigmatism. Otherwise no congenital abnormality detected.
77	Two to four months.	Cleft palate. Otherwise no congenital abnormality detected.
78	Early months.	No congenital abnormality detected.
79	Early months.	Grade III deafness. Otherwise no congenital abnormality detected. Attending School for Blind and Deaf.
80	Five months.	Mongoloid type. Cannot be taught. Hearing and vision normal.
81, 82, 83, 84, 85, 86, 87, 88, 89	Five months.	No congenital abnormality detected.
90	Early stages.	Mentally poor. Left eye, congenital cataract. Right eye, myopic. Astigmatism, pulmonary murmur.
91	Five months.	Grade IIB deafness.
92	Five and a half months.	No congenital abnormality detected.
93, 94, 95, 96	Six months.	No congenital abnormality detected.
97	Four and a half months.	No congenital abnormality detected.
98	Early months.	No congenital abnormality detected.
99	Five to six months.	No congenital abnormality detected.
100	Six months.	Refractive error, right eye. Otherwise no congenital abnormality detected.
101	Four and a half months.	No congenital abnormality detected.
102, 103	Five to six months.	No congenital abnormality detected.
104	Six to seven months.	No congenital abnormality detected.
105	Indefinite.	No congenital abnormality detected.
106	Eight months.	No congenital abnormality detected.
107	Seven months.	Mentally deficient. Mongoloid type. Heart and hearing normal. Strabismus.
108, 109, 110	Seven months.	No congenital abnormality detected.
111	Seven months.	High frequency deafness, left ear.
112	Six to seven months.	Grade III deafness. Attending School for Blind and Deaf. No other congenital abnormality detected.
113, 114, 115, 116, 117	Six months.	No congenital abnormality detected.
118	Seven and a half months.	No congenital abnormality detected.
119	Indefinite.	No congenital abnormality detected.
120	Later stages.	No congenital abnormality detected.
121	Eight and a half months.	No congenital abnormality detected.
122	Indefinite.	No congenital abnormality detected.
123	Indefinite.	No congenital abnormality detected.
124	Indefinite.	No congenital abnormality detected.
125	Seven to eight months.	Congenital cataract. Otherwise no congenital abnormality detected.
126	Indefinite.	Grade III deafness. Systolic murmur all areas. Heart enlarged. Attending School for Blind and Deaf.
127	Indefinite.	Grade III deafness. Otherwise no congenital abnormality detected. Attending School for Blind and Deaf.
128	Indefinite.	No congenital abnormality detected.
129	Three to four months.	Grade III deafness. Systolic murmur, pulmonary and aortic areas.

[1] *Vide infra*, explanation of grades of deafness.

children were said to be mentally deficient and two were stated to have congenital heart disease. It is quite possible that more cardiac abnormalities would be found on clinical examination. Among the 36 children with abnormalities, 29 mothers stated that they had suffered from rubella at some time in the first four months of pregnancy, three were uncertain of the stage of pregnancy and only four stated that they had suffered from rubella after the fourth month.

Discussion.

Until Gregg[2] published his work in 1941, rubella was generally regarded as a nuisance rather than as a disease. In few countries was the disease notifiable, and those health authorities who regarded it as such were considering deleting it from their list of notifiable diseases. It is noteworthy that the first work on this problem was done by Australians. The American, Prendergast,[3] suggests that the condition originated in Australia and spread to the Pacific coast of America. Following Gregg's publication, further work was done by Swan,[4][5][6] of South Australia, by the committee appointed by the Director-General of Public Health in New South Wales,[7] by Evans,[8][9] by Winterbotham[10] and by others. Several articles have appeared in American journals, and the subject has also caught attention in Britain and Holland.[11]

Table II.

Abnormality.	Number of Patients Showing this Particular Abnormality.
Grade III deafness (deaf mutism)	27
Grade IIB deafness	2
Grade IIA deafness	1
Grade I deafness	4
High frequency deafness (hearing for speech normal)	3
Total with hearing defects	37
Congenital heart disease	10
Mental deficiency	6
Retarded mentally, but not definitely mentally deficient	1 } 7
Cataract	2
Cleft palate	1
Malformed ear lobe	1
Grand total	58

Doubt that rubella was the disease to be blamed for these abnormalities was expressed by Gregg in his original paper. He suggested that the real disease could be due to a streptococcus, and the rash a toxic erythema.

Swan pointed out that at the same time as the rubella epidemic an acute respiratory infection was rife amongst army personnel, and was known under various names, such as Ingleburn, Puckapunyal and Woodside throat. This was also recorded in the "Current Comment" columns in The Medical Journal of Australia about that time.[12] However, cases occurred before this sore throat epidemic, and are still occurring, in which the disease is diagnosed as rubella. Although the case has not been proved experimentally, the consensus of opinion points to rubella as the disease causing these abnormalities.

In drawing any conclusions from the results of the present survey, one must bear in mind the following facts. Seven years had elapsed between the contraction of the disease and the time when the *questionnaire* was answered. In most instances it is believed that the disease was self-diagnosed. It is pointed out, however, that unless the *questionnaire* was answered definitely, it was not included in the results.

Geographical Distribution.

It must not be presumed that because children attended a particular school at the time when the survey was made, their mothers were living in that area at the time of the epidemic. From those cases in which it was possible to ascertain the place where the disease was contracted, it is

learnt that the epidemic was prevalent along the whole of the coast of Queensland. After Brisbane, the heaviest incidence occurred on the Darling Downs and the far north. There were many cases in Toowoomba and Innisfail. There was apparently little spread to western Queensland. One private practitioner volunteered the information that there has been no rubella in Charleville for twenty years.

The Percentage of Abnormalities.

It is difficult to assess accurately the risk that a mother suffering from rubella during pregnancy will produce a child with an abnormality. In this survey all children had reached six years of age. Evidence was gathered

Table III.

Abnormality.	Number of Cases.
Grade I deafness	1
High frequency deafness (hearing for speech normal)	3
Congenital heart disease	2
Eye defects (including strabismus, myopia, amblyopia and astigmatism—there were no cataracts)	12

which showed that some mothers had suffered miscarriages and some had had stillborn infants after having suffered from rubella in pregnancy. The rubella may or may not have been the cause.

In Gregg's series of 78 cases, 15 children died before they reached the age of twelve months. Bearing these points in mind, the reader is referred to the figures presented above.

In Brisbane, 28 children out of 77 cases, whose mothers had rubella in the first four months of pregnancy, had serious abnormalities, and a further 11 had a minor abnormality; thus 39 of 77 children had abnormalities from maternal rubella contracted during the first four months of pregnancy.

The Period of Pregnancy in Relation to the Abnormality.

The Americans Aycock and Ingalls[13] give definite periods of pregnancy, during which contraction of rubella by the mother is followed by a particular abnormality in the infant.

(No effort was made to do this in the present work, because the mother's memory would be the only record of the exact week of the onset of the exanthem. In fact, it may be thought that after seven years undue credence is being given to the accuracy of the replies in quoting months, as has been done in most cases.) Their findings in this regard are as shown in Table IV.

Table IV.

Stage of Pregnancy.	Likely Abnormality.
Six weeks	Cataract.
Nine weeks	Deafness.
Five to ten weeks	Cardiac abnormality.
Six to nine weeks	Deformed teeth.

However, it is pointed out, firstly, that the figures presented add to the evidence of other workers that the risk is greatest during the early months of pregnancy—one might add the first four months—and secondly, when a combination of abnormalities is found, it is hard to lay down that rubella in a certain week of pregnancy will be followed by one particular abnormality.

The Abnormalities.

The most frequent abnormality found was deafness; then followed congenital heart disease, mental deficiency and cataracts.

Dr. E. O. Marks, ophthalmologist, who has examined the eyes of the children attending the School for the Blind and Deaf, reports the occurrence of a pigmentary abnormality in the fundi of these "rubella" children. Their vision is unaffected.

Mr. E. W. Haenke, Chief Dental Inspector, School Health Services, examined 24 of the children attending the School for the Blind and Deaf with a maternal rubella history, and reported as follows:

> As recorded at the individual mouth inspections, caries was classified as free in 9 cases, mild in 9 and moderately severe in 6 of the number examined.
>
> As such findings show a betterment rather than a decline when compared with recordings taken from corresponding cross-sections of the community, they do not lend support to the idea that maternal rubella may have exercised an adverse influence on the incidence of dental caries in this group of children.
>
> Further dissection of the respective chartings, however, did unfold some findings of professional significance which, it is thought, could have their origin in the upsetting influence of rubella.
>
> They were:
>
> (*a*) Congenital absence of isolated deciduous teeth.
> (*b*) Hypoplasia.
> (*c*) Retardation in tooth eruption.
>
> Congenital absence of teeth. In one child the temporary dentition showed evidence of the congenital absence of the upper left deciduous lateral incisor. The abnormality was detected by clinical observation. It was not confirmed by an X-ray examination.
>
> Hypoplasia. Dental hypoplasia was observed in 8 per cent. of the mouths examined. In each case the condition was mild and was localized to several teeth. Such hypoplasia frequency is greater than that recorded at regular school inspections of other groups of children of corresponding ages.
>
> Retarded eruption. Whilst investigations regarding rubella which have been carried out by different research workers have shown, as would be expected, that variations from normalcy in the eruption of the teeth are associated principally with the deciduous dentition, it is of interest to mention that in this small group of cases with a 1941 birth-year index, there was a definite retardation in the eruption of the 6 year molars in 19 per cent. of the total cases.
>
> Such departure from normalcy in the eruption of this group of teeth might not be regarded as pronounced according to some tooth-eruption tables, but, nevertheless, it has been deemed sufficiently definite and constant as to warrant a reference to its occurrence.

Experimental Work.

It has not yet been proved experimentally that rubella in pregnancy produces congenital abnormalities, but it is worth quoting here what has been done in isolating the organism and in the production of the disease in laboratory animals. The following extract from Aycock and Ingalls's article is worth repeating here.

> The infectious agent is a filterable virus and filtered naso-pharyngeal washings give rise to the infection in susceptible beings upon subcutaneous injection. The virus particles are demonstrable by fluorescent microscopy in nasal and pharyngeal washings in blood and in blister fluid of injected patients. Habel[14] has inoculated macacus rhesus monkeys subcutaneously, intraperitoneally, intranasally and intravenously, with the subsequent development of a mild infection characterized by a leukopenia, relative lymphocytosis and fever (average 0·5°) followed by a "very light scattered macular rash on the face, abdomen and thighs, starting about the 11th day and followed by desquamation". He was also successful in culturing the virus on the chorio-allantoic membrane of the chick embryo in 2 of 4 attempts.

Conclusion.

Despite the lack of experimental evidence, there is sufficient clinical evidence in this and other reports to show the grave risk of exposure of the pregnant woman to rubella. In conclusion, two practical points are raised: (i) What can be done to prevent these abnormalities? (ii) What can be done to treat and educate the children in which the abnormalities occur?

Prevention.

The following measures and certain criticisms are presented.

1. Rubella might be added to the list of notifiable diseases.
2. Deliberate exposure of all girls to the disease might be practised. This would depend on the availability of the disease, but otherwise seems feasible.
3. Inoculation with rubella might be developed. Suitable techniques are not yet available.
4. Exposed pregnant women might be treated with immune globulin. The efficacy of this measure has not yet been established.
5. Pregnancy might be terminated if rubella had been contracted in the first four months—a certain method, but its justification is debatable.
6. A warning might be given to all pregnant women, or women thought to be pregnant—the diagnosis in the danger months is not always definite—to avoid exposure. There is no bar to its implementation.

Treatment and Education.

Treatment of these handicapped children is the province of the medical specialist and will not be discussed here. Education of these children is in the hands of a specialist too—the educational specialist. There are two aspects to be considered: (*a*) the education of the mentally deficient; (*b*) the education of the deaf child. (The child with cardiac abnormalities and no other defect needs no special provision other than those provided in our State schools.)

In Queensland, the mentally deficient, including those in the rubella group, may be educated at the special Opportunity School at Dutton Park and at Opportunity grades attached to several of the larger schools. None of these, however, provide boarding accommodation, and it is suggested that there is a need for a resident school for these children.

The education of the deaf child of the prescribed age is admirably catered for at the School for the Blind and Deaf, which is a resident institution; but owing to the increase from the rubella group—from the two epidemics of 1938 and 1940 there were approximately 60 admissions—the accommodation is taxed to the utmost.

Proposed temporary increase of accommodation will be immediately filled by a waiting list—it is a deplorable fact that there is a waiting list. Many of the children at present enrolled are from North Queensland, and from the returns of this survey there are several more deaf children attending ordinary schools in the Innisfail and Cairns areas.

It is therefore suggested that at least a temporary school for the deaf be established in the north.

In addition to the need for extra accommodation for children of the prescribed age—children may be admitted at the School for the Blind and Deaf at four years—it is suggested that provision be made either at the present school or elsewhere for younger deaf children. Education of these children should be commenced as soon as deafness is diagnosed.

In England, under the *Education Act* of 1944,[15] "it is the duty of the Local Education Authority to provide education for a deaf child from the age of two if the parents desire it. The age at which education becomes compulsory is five". Children of this earlier age could be traced by the toddlers' clinics conducted by the Child and Maternal Welfare Department, and by private practitioners.

Acknowledgement.

I am indebted to Dr. A. Fryberg, Director-General of Health and Medical Services, for permission to publish this report.

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OBSERVATIONS ON THE USE OF MIXTURES OF PROTAMINE ZINC AND REGULAR INSULINS IN THE MANAGEMENT OF DIABETES MELLITUS.

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THE establishment of satisfactory control of severe *diabetes mellitus* presents a problem which can tax the ingenuity and resourcefulness of any physician. Despite the enormous advances which have been made in the management of this disease in the past twenty-five years, the limitations of treatment with insulin are apparent to anyone with experience in handling large numbers of such cases.

Soon after the introduction of regular insulin to practice it was found that the responses of patients varied considerably from one to the other, and two main difficulties were encountered. The first was the impossibility, in some instances, of keeping the blood sugar level within normal limits during the day without sudden variations which produced hypoglycæmia. The second was that of maintaining a normal blood sugar level throughout the night. The vast majority of patients with mild or moderately severe diabetes were managed with two or three injections of regular insulin given before meals; however, in some instances this failed to achieve adequate control. So for many years attempts were made to produce some preparation of insulin which would lessen or overcome these troubles. Leyton suspended crystalline insulin in oil, and Clausen suggested the use of a vasoconstrictor injected simultaneously with insulin. Neither of these methods achieved any success, and it was not until 1936 that Hagedorn[1] made a real advance. He discovered that insulin combined with a protamine (a simple protein obtained from the roe of a fish) formed a relatively insoluble compound which was absorbed slowly. This substance appeared commercially as "Insulin Retard". Very shortly afterwards Scott[2] showed that the addition of small quantities of zinc to this protamine combination produced a compound which was more stable and which

had a more prolonged action. Protamine zinc insulin, now universally accepted, represents the first satisfactory attempt to provide an insulin of slower and more even effect. The relative differences between the times of action of regular insulin and of protamine zinc insulin can be summarized thus: over 70% of the total effect of a dose of regular insulin is obtained within eight hours of injection, while only 20% of the total effect of a dose of protamine zinc insulin is obtained in the same period. Further, approximately 20% of the total dose of regular insulin is effective in the period between twelve and twenty-four hours after injection, while over 50% of a dose of protamine zinc insulin is active during this time.

Protamine zinc insulin has proved to be of great value in managing many of the mildly affected patients whose diabetes previously was controlled only by two injections of regular insulin, and particularly those in whom a sharp rise of blood sugar level occurred during the night. Once again, however, this form of insulin has been found to have its limitations, particularly in controlling hyperglycæmia during the daylight hours. A dose sufficient to maintain a normal blood sugar level throughout the day is frequently responsible for hypoglycæmic reactions in the early hours of the morning. Another compound, globin insulin, the effects of which are intermediate in time between those of regular insulin and protamine zinc insulin, although theoretically more useful, has also been found to be incapable of controlling the blood sugar throughout twenty-four hours in many cases of average severity.

It is not surprising, therefore, that attempts have been made to combine the effects of rapidly acting and of slowly acting insulins in order to manage difficult cases. At first regular insulin and protamine zinc insulin were injected simultaneously, but at different sites, and in an earlier communication I discussed the usefulness of this procedure.[3] More recently, the two forms of insulin have been combined and administered as one injection. It must be realized that with any mixture of protamine zinc insulin and regular insulin the effects produced are likely to be appreciably different from those obtained by similar doses given as separate injections at different sites. This was clearly demonstrated by Hildebrand and Rynearson.[4] Some protamine zinc insulin preparations contain an excess of protamine, and when any regular insulin is added it will be altered, if not exactly to protamine zinc insulin, to a compound which has a delayed action.[5][6] The exact effect of such a mixture will obviously bear some relation to the ratio between the amounts of the fast and slow components.[7] As a rough assumption Lawrence[8] has postulated that X units of protamine zinc insulin can adsorb and change $\frac{X}{2}$ units of soluble insulin.

Considerable interest has been shown in the United States of America in the use of these mixtures, and an extensive literature has accumulated in the past five years on the results obtained by various observers. In general, the American work has been concerned with the determination of an optimal ratio between fast and slowly acting insulins in an attempt to produce one mixture which could be used in the treatment of all patients. The different proportions suggested by clinics of high repute[4][6][10][11] give clear evidence that no one fixed mixture is possible. This must not be regarded in any way as suggesting that mixtures are unsatisfactory. On the contrary, in skilful hands they represent the best attempt so far made to provide means of control in difficult cases.

The purpose of this communication is to record my experience with variable mixtures of protamine zinc insulin and regular insulin in a series of 100 patients who have been treated in the past twelve months. Fifty-nine of these patients had not received insulin previously, while 41 had been taking regular or protamine zinc insulin alone. No attempt was made to work with any fixed ratio of insulin mixture. Each patient was considered individually, and in every instance an effort was made to arrange such a combination of the two insulins as would suit the particular requirements of the case.

The general management of those patients who had not received insulin previously followed conventional lines, of which details have been given in an earlier communication.[12] Without exception they were admitted to hospital for the initial period of stabilization, and an appropriate diet and an insulin dose were worked out while the patient was under close observation.

In every case the insulin mixture was made immediately prior to injection by combining the two insulins according to the following technique.

Under appropriately aseptic conditions a small quantity of air is injected into the protamine zinc insulin bottle and the needle is then withdrawn. No insulin is taken from the bottle at this juncture. The needle is then inserted into the regular insulin bottle, an appropriate amount of air is injected and the dose of regular insulin is drawn into the syringe. The needle is then reinserted into the protamine zinc insulin bottle and the dose is withdrawn. A small bubble of air is sucked into the syringe, and the syringe, held horizontally, is rolled between the fingers, the air bubble being used to assist in mixing. The air bubble is then expelled and the mixture is ready for injection.

Strict adherence to this technique is desirable, and it is essential, to avoid confusion, that insulins of the same strength be used. It is also important to use the same brand of insulin continuously, as the protamine content of different preparations varies slightly. It matters little if a minim or two of regular insulin escapes into the protamine zinc insulin bottle; but a few minims of protamine zinc insulin inadvertently entering the regular insulin bottle will materially alter the composition of its entire contents.

Each patient was taught this technique under close supervision, and injections were given in the presence of a trained observer until proficiency was acquired. The stabilization of patients in this group proceeded according to the following plan. The initial dose of insulin was made up of equal parts of protamine zinc insulin and regular insulin. The total daily dosage was decided by such factors as the age of the patient, the duration and severity of symptoms, the height of the fasting blood sugar level, the presence or absence of ketonuria, and the estimated caloric requirements of the patient. In every case the injection of insulin was made twenty minutes before the morning meal. If after a few days no appreciable alteration had occurred in the glycosuria at any period of the day, the total dosage of the equal mixture was raised until the urine began to clear. Thereafter an increase of one or other of the components was made as follows. If greater insulin action appeared necessary between 8 a.m. and 6 p.m., the dose of regular insulin was raised. If, on the other hand, further insulin activity was needed throughout the night, the dose of protamine zinc insulin was raised. This proved to be a useful guide in establishing the appropriate proportions of the two insulins. As stability throughout the twenty-four hours was approached, estimations of the blood sugar level in the fasting state and at intervals throughout the day gave exact evidence of the individual patient's response to the mixture determined for him. Minor adjustments of diet and the placing of intermediate feedings were made as occasion demanded.

An example of the stabilization of a patient with untreated diabetes is shown in detail in Table I.

The patient was a male, aged fifteen years, who had noticed thirst and frequency of micturition for four weeks prior to seeking medical attention. His mother and grandfather had both suffered from diabetes. The past history of the boy revealed nothing of significance. No abnormality was found on physical examination other than an obvious loss of weight (approximately eight pounds in four weeks). His urine was loaded with sugar and also contained acetone and aceto-acetic acid. A glucose tolerance test was performed with 50 grammes of glucose given by mouth, and yielded the following results: fasting blood sugar level, 0·24%; blood sugar level half an hour after ingestion of glucose, 0·26%; one hour after ingestion of glucose, 0·32%; one and a half hours after ingestion of glucose, 0·35%; two hours after ingestion of glucose, 0·41%. He was admitted to hospital at once, and was discharged sixteen days later with the diabetic condition adequately controlled by appropriate diet and insulin dosage. His subsequent progress has been steady and uneventful.

TABLE I.

Day.	Urine Examination (Sugar Content).				Insulin. (Units.)	Weight.	Remarks.
	7 a.m.	10 a.m.	2 p.m.	8 p.m.			
1			2·0%	2·0%	R[1] 20 at 5 p.m.		
2	1·0%	1·0%	1·0%	2·0%	PZ[1] 20 at 8 a.m. R 20	8 stone 6 pounds	Diet 2525 Calories (carbohydrate 246, protein 104, fat 125).
3	0·6%	0·6%	0·2%	1·0%	PZ 20 R 20		(a)
4	2·0%	1·0%	0·2%	0·6%	PZ 20 R 20		
5	0·2%	2·0%	2·0%	0·0	PZ 20 R 20	8 stone 12 pounds	
6	0·2%	1·0%	2·0%	0·2%	PZ 10 R 20		(b)
7	0·6%	1·0%	2·0%	2·0%	PZ 20 R 20		
8	2·0%	2·0%	1·0%	1·0%	PZ 20 R 20	8 stone 13 pounds	
9	2·0%	1·0%	0·2%	1·0%	PZ 16 R 32		(c)
10	0·0	0·6%	0·2%	0·6%	PZ 16 R 32		
11	0·0	1·0%	0·2%	0·2%	PZ 16 R 32	9 stone 1 pound	Slight reaction 11.45 a.m.
12	0·0	0·2%	0·2%	0·2%	PZ 12 R 28		(d)
13	0·0	0·6%	0·2%	0·2%	PZ 12 R 28		
14	0·0	0·2%	0·2%	0·0	PZ 12 R 24	9 stone 1 pound	
15	0·0	0·0	0·0	0·0	PZ 12 R 24		
16	0·0	0·0	0·2%	0·2%	PZ 12 R 20		(e)

[1] R=regular insulin; PZ=protamine zinc insulin. (a) Initial insulin dose determined by factors described in text. (b) Premature reduction of protamine zinc insulin in response to findings on fifth day. (c) Increase in dosage and alteration in ratio because of apparent failure of previous pattern. (d) Reduction of dosage in response to previous day's findings. (e) Reduction of dosage in anticipation of increased exercise after discharge from hospital.

The 59 patients considered in this group varied in age from three to seventy-four years; 32 were females and 27 were males. Nineteen of these patients were satisfactorily controlled with a mixture of equal parts of the two insulins.

In twenty-four cases the dosage of protamine zinc insulin exceeded that of regular insulin, the greatest variation being three parts to one. In the remaining sixteen cases the dosage of regular insulin exceeded that of protamine zinc insulin, the greatest variation being two and a half parts to one. The insulin requirements in the whole group varied from a minimum of 12 units per day to a maximum of 120 units per day. The average total daily dosage was 37·5 units and the averages of the protamine zinc insulin and regular insulin doses were 20·3 and 17·2 units respectively.

In the majority of cases stabilization proceeded uneventfully and was achieved without difficulty in no greater period of time than would have been required if either type of insulin had been used alone. Hypoglycæmic reactions were infrequent, and when they occurred were mild and easily controlled. The majority occurred between 4 p.m. and 6 p.m., and were subsequently averted by a small feeding between 4 p.m. and 4.30 p.m. In a few instances reactions occurred in the later part of the morning or during the night. An alteration in the proportions of the slow and fast insulins usually served to correct this. On a few occasions a persistent headache lasting for some hours was found to be associated with low blood sugar values. A small reduction of the total dosage abolished this symptom.

In two cases (both patients were females) stability was attained only after a period of many weeks. In neither instance was the diabetic condition unusually severe, and the final insulin dosages were 44 and 38 units per day respectively. In both cases considerable temperamental instability was present, and it is more than probable that this was responsible.

In the remainder of the cases the average length of time from the commencement of insulin treatment to the attainment of satisfactory control was between two and three weeks.

Included in this series were four children, aged respectively three, four, six and thirteen years. No difficulty was experienced in their management, and realizing the desirability of one daily injection for the child diabetic, I am satisfied that better control was accomplished than could have been attained by one injection of either insulin alone.

Many of the 41 patients who were already taking insulin were changed over to mixed insulin without a period in hospital. The cases in this group fell rather simply into two categories: those in which the diabetic condition was satisfactorily controlled either by regular or by protamine zinc insulin, and those in which for various reasons control was unsatisfactory. If a patient was considered sufficiently intelligent and trustworthy, the daily reporting of the results of urine examinations together with the results of appropriate blood sugar estimations formed an adequate basis for the adjustment of dosage until stability was regained. If a patient was not considered capable of adjusting himself without close supervision, he was admitted to hospital for a short period. With one exception—a man who, from lack of intelligence, was found incapable of mixing insulins with any degree of accuracy—all patients in this series have been as well controlled as formerly, and in many instances better controlled.

Twenty-seven patients had previously taken two injections of regular insulin each day. The plan of their transfer to a mixture followed these lines. If the patient was taking X units of soluble insulin in the morning and Y units in the evening, on the first day of the transfer he took X units of regular insulin mixed with $\frac{Y}{2}$ units of protamine zinc insulin in the morning and $\frac{Y}{2}$ units of regular insulin in the evening. On the second day he received X units of regular insulin *plus* Y units of protamine zinc insulin as one injection in the morning, and no further insulin was given that day. In some instances, and particularly where the dosage was high, a more gradual change was made extending over three or four days. Immediately after transfer the total dosage was left unaltered for several days, and then the components were modified as indicated by the results of urine tests and blood sugar estimations. Persistent glycosuria in the evenings or overnight necessitated a rise in the protamine zinc insulin dose, whilst glycosuria throughout

the day pointed to the need for an increase in the regular insulin dose. No difficulty was found in rapidly transferring these patients from one *régime* to another. It is interesting to note that 12 patients previously regarded as adequately controlled showed an average reduction of 5·3 units in their daily requirements when changed to a mixture.

The remaining 15 patients were regarded as being inadequately controlled. In some instances this was due to lack of supervision during the war years or to carelessness, but in others it was due to the fundamental difficulty of controlling the diabetes with regular insulin. Case XXXVII is an interesting example of this.

R.F., a male patient, aged forty-three years, had suffered from diabetes for fourteen years. Ten years ago he contracted pulmonary tuberculosis, which responded to a period of sanatorium treatment, and the pulmonary lesion has been regarded as healed for the past seven years. For many years his diabetic condition proved difficult to control, and he suffered from severe hypoglycæmic reactions which occurred without warning. Throughout his treatment various patterns of dosage with both fast and slow insulins were tried, but never with very satisfactory results. Figure I shows in graphic form the pattern of his blood sugar levels

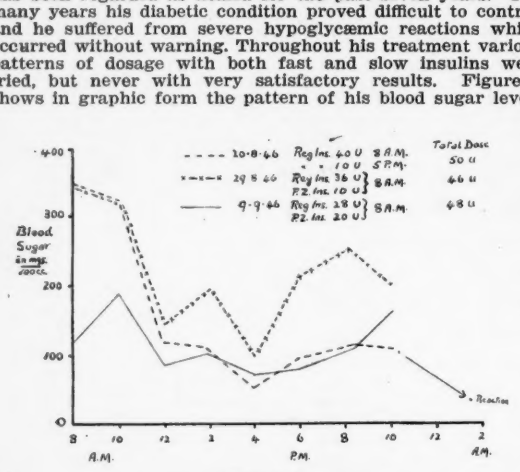
10·8·46 Reg Ins. 40 U 8 A.M. + 10 U 5 P.M. Total Dose 50 u
29·8·46 Reg Ins. 36 U PZ Ins. 10 U 8 A.M. 46 u
9·9·46 Reg Ins. 28 U PZ Ins. 20 U 8 A.M. 48 u
Blood Sugar in mgs. per 100 cc.
400
300
200
100
0
Reaction
8 10 12 1 4 6 8 10 12 2
A.M. P.M. A.M.

Figure I.

before and after his transfer to a mixed injection once a day. It is of particular interest to note that with the more even action of the mixture stability was attained on a smaller dose. He has remained well for the past nine months with only an occasional hypoglycæmic reaction.

Thirteen patients had previously taken one injection of protamine zinc insulin each day. No uniform procedure for transfer was adopted, as it was found necessary to consider each case individually. Six patients were regarded as being reasonably controlled; but in view of nocturnal reactions it was thought desirable to change to a mixture. Case LXIV is an example of this.

D.R., a female patient, aged twenty-eight years, who had been a diabetic for ten years, had been taking forty units of protamine zinc insulin each morning. At intervals of two or three months she was prone to unexpected and severe insulin reactions between 4 a.m. and 8 a.m. Any decrease in the dosage of protamine zinc insulin resulted in hyperglycæmia and glycosuria during the day. She was changed in one move to a mixture of 32 units of protamine zinc insulin and eight units of regular insulin, with no disturbance of control. No further insulin reactions occurred in the early morning; but it is interesting to record that feelings of impending hypoglycæmia, confirmed biochemically, occurred occasionally at 6.30 p.m., necessitating a feeding in the mid-afternoon.

This observation is of particular interest. As the addition of small amounts of regular insulin to protamine zinc insulin usually results in conversion of the regular fraction to a compound with similar action to that of protamine zinc insulin, one would not expect such an effect.

All six patients were more satisfactorily stabilized on the mixture than on protamine zinc insulin alone, although on the average there was no alteration in the total daily dose.

Seven patients were regarded as inadequately controlled, in that glycosuria and hyperglycæmia occurred during the day, while the fasting urine was sugar-free and the fasting blood sugar level was within normal limits. Any attempt to increase the protamine zinc insulin dose failed to check the daytime hyperglycæmia, but resulted in nocturnal hypoglycæmia. Each patient was transferred to a mixture by gradual addition of regular insulin and reduction of the protamine zinc insulin, the daily dosage being left unchanged until such time as it was evident that stabilization had occurred or that an increase in the total dose was necessary.

One patient had been taking one daily injection of globin insulin which did not maintain appropriate control of her condition. With no alteration in total dosage, she was changed with entirely satisfactory results to a mixture of a ratio of two units of protamine zinc insulin to one unit of regular insulin.

Of the 41 patients transferred from one or other type of insulin to a mixture, the maximum dosage required in any one case was 200 units per day (110 units of protamine zinc insulin to 90 units of regular insulin). (The patient was a boy of nineteen years, a cabinet-maker, who has suffered from diabetes for eight years and is now in excellent health.) The smallest dose in this series was 22 units each day (eight of protamine zinc insulin to fourteen of regular insulin), taken by a woman, aged fifty-three years, who had had diabetes for seven years. The youngest patient in this group was a female, aged three years, who had been a diabetic for eighteen months (dosage, 56 units—28 of protamine zinc insulin and 28 of regular insulin); the eldest was a female, aged sixty-seven years, who had suffered from diabetes for eighteen years (dosage, 96 units—48 of protamine zinc insulin and 48 of regular insulin).

The relative proportions of the two insulins in the mixtures used have varied from three parts of regular insulin to one part of protamine zinc insulin, to one part of regular insulin to four parts of protamine zinc insulin.

Summary.

It is apparent from a study of the cases described in this paper that the use of mixed insulins is of considerable value in the management of *diabetes mellitus*. It is difficult to lay down rules for use in any one case. While it is possible to give in outline a scheme for dealing with a new patient, every case must be considered individually and the ultimate insulin dosage must be arrived at by a process of trial and error. While this involves a period of close observation in the first few weeks of treatment, the results in my experience more than justify this procedure. While in this series no attempt has been made to fit each patient to a fixed mixture of insulins, it would seem from the wide variation found in the components of the mixture that it is unlikely that any fixed mixture could be as satisfactory as one determined by the needs of the individual. The flexibility of adjustable doses of fast and slow components has proved satisfactory in the handling of those patients who are prone to sudden and unexpected hypoglycæmia with regular insulin. Another group in which mixtures have proved their worth includes those patients whose diurnal hyperglycæmia cannot be controlled with protamine zinc insulin without the production of severe hypoglycæmia during the night.

From my present limited experience it would seem that extemporaneous mixtures provide a most satisfactory method of prescribing insulin, as they permit a finer adjustment to the patient's needs than is possible by any single preparation of insulin which is available so far. Lastly, those patients who have been freed from the necessity of evening injections experience an extraordinary freedom of life, which is reflected in their sense of well-being and in their outlook on the disease.

Conclusions.

1. The use of extemporaneous mixtures of protamine zinc insulin and regular insulin in the treatment of 100 diabetic patients is described.

2. **No fixed mixture is applicable to all patients, and it is emphasized that individual requirements may necessitate wide variations in either component.**

3. **A single injection of an appropriate mixture has been found adequate to control the diabetic state as well as or better than any other pattern of insulin dosage.**

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THE SYNDROME OF CHRONIC HYPERTENSION, FIBROSITIS AND TONSILLITIS.[1]

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ON June 16, 1941, I was called to see Master F.K., aged twelve years, suffering from severe dyspnœa of recent onset, with hypertension, œdema, an apex beat in the axilla and heavy albuminuria. On my arrival he was being supported by relatives with his head partly through an open window in order "to give him more air". His illness had been preceded by mild acute pharyngitis. I thought that he would die. He was admitted to Hornsby District Hospital under the care of Dr. F. A. E. Lawes, and he recovered.

Such cases are not common, but they form a well recognized type, grouped by Ellis[(1)] as type 1 nephritis. Ellis discovered a history of infection in 84% of his cases of type 1 nephritis—usually tonsillitis or sore throat or "cold". It is probable that if his patients had been examined before the nephritis occurred, the infection would have been revealed in the other 16%. This probability will be appreciated by those who, like myself, have seen children suffering from repeated short bouts of listlessness, perhaps with vomiting, described by the mother as "bilious attacks". The attacks are mild and are sometimes thought to be just a passing tiredness. Sometimes the mother reports that she has found the temperature elevated (100° to 102° F.). These febrile attacks remain of unknown origin until the throat is examined during one of them. The acute infection in the tonsils is then discovered. There is another group of children with acute tonsillitis in whom listlessness is the only symptom, and the mother notices a visible enlargement of the tonsillar lymph glands. Thus, infection may produce in different patients an evenly graded series of symptoms, from the minor, which often pass unnoticed or unrecognized as the symptoms of disease, to the more pronounced and classical clinical pictures. Acute tonsillar infection may run its course without producing any symptoms referable to the throat. These facts, together with the well-known forgetfulness of patients for even recent minor incidents, make it probable that there was much more infection among Ellis's patients than could be revealed by a retrospective analysis.

The case of Master F.K., and the above considerations, naturally raised the question whether a chronic latent infection in the tonsils may not be one of the factors causing chronic hypertension. Certain objections are raised to such an assumption. But the objections are not substantial. Firstly, it is said, patients with chronic hypertension may not give a history of sore throat. (But chronic tonsillitis does not, as a rule, cause sore throat.) Secondly, many patients suffer from chronic tonsillitis but do not develop hypertension. (But many children suffer from throat infections and do not develop type 1 nephritis.) Thirdly, heredity is an important factor in hypertension. How does this fit in with tonsillitis? (The lodgement of bacteria and consequent infection in the tonsils are a universal and normal part of our struggle for existence. Perhaps an hereditary weakness in certain tissues is one of the factors which determine hypertension in some and not in others. In the same way, the upright posture is a normal part of our existence, but only some of us develop varicose veins. We are all exposed to the strains and stresses of modern life, but only some of us acquire a duodenal ulcer. We are all exposed to allergens, but only some of us suffer from asthma or migraine. Hereditary weaknesses in tissues or tissue fluids are among the factors which help select the victims.) Fourthly, many patients with hypertension pass no albumin or casts in their urine. (But the Goldblatt kidney, in which renal ischæmia is produced by a metal clip on the renal artery, causes hypertension, and may, at the same time, secrete a urine which contains no albumin or casts. In fact, the kidney may be indistinguishable clinically and histologically from the normal kidney. For this information I am indebted to Dr. F. B. Byrom.)

Such considerations led me, during the last three years of my general practice, to scrutinize closely my patients with chronic hypertension for evidence of tonsillitis, and to advise patients to submit to tonsillectomy if such evidence was forthcoming. Although the material which I have collected is incomplete and inconclusive, I would like to present it now, because opportunities for prolonged observation are not now readily available to me, and the observations, although incomplete, suggest that the question should still be pursued. The patients whom I now observe for any considerable length of time have been submitted to sympathectomy and they are thus not so suitable for observation as those others who are cared for entirely by their private medical practitioner.

The Indications for Tonsillectomy.

The following are indications for tonsillectomy.

A History of Fibrositis.

The history of rheumatism or fibrositis, which I take to be of importance in evaluating the rôle of the tonsils, must be specially inquired into by the clinician, and leading questions about "lumbago", "neuritis", pains and aches and stiffness in joints, must be skilfully used. Such symptoms are not volunteered by the patient, who has no immediate recollection of them, or perhaps feels that they are minor and irrelevant. They are often forced into the background by more severe symptoms. Sometimes, at a first consultation, patients will deny any "rheumatism" but later come back with a different story after they have thought it over and talked it over. Two recent examples, referred by Dr. C. G. Champion for possible surgical treatment, are as follows.

Mrs. M.E.L., aged forty-eight years, came on October 1, 1947, complaining of headache and of giddiness. She said

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on October 23, 1947, at Saint Vincent's Hospital.

nothing about rheumatism until directly asked. Five years earlier she had been treated for rheumatism at South Sydney Hospital. Later she had had pains in the left shoulder, right wrist and elsewhere. One year prior to coming to me she had been treated for acute rheumatism. The tonsils had been "removed" twenty years earlier. Examination revealed large infected tonsil remnants.

Mr. J.L.S., aged fifty-three years, a solicitor, came on October 15, 1947, complaining of a "dopey" feeling and of headaches. I asked him about "rheumatism", and he replied as follows: "Dr. Champion asked me about that, and I told him that I never had rheumatism. But since then I remembered that some years ago I was in bed for two weeks, and under the doctor's care, on account of lumbago. Some months ago I noticed rheumatism in the right shoulder while I was playing tennis." I also learned that he took Glauber salts regularly because someone had said that they are good for rheumatism, and he always carried tablets of potassium chlorate "to prevent coughing in court". His tonsils were grossly infected. They had been "removed" in childhood.

Findings on Inspection.

On inspection the anterior pillar of the fauces may look more red than the adjoining palate. But often the tonsils show no evidence of abnormality on first inspection. This must not be taken as evidence of normality.

Findings on Pressure.

If the tongue is held down by a spatula, and a second spatula is used to press upon the anterior pillar and evert the tonsil, the tonsil may look much larger than was at first thought, red and succulent as if œdematous, and yellow fluid may be expressed from it. I have taken such evidence as weighing heavily in favour of a tonsillar infection.

I should like to stress that a normal superficial appearance of the tonsils does not prove their innocence of an ætiological rôle in hypertension. Dr. Lance Corner has told me of some striking examples of obstinate acute nephritis in young soldiers, which cleared up after the removal of tonsils which looked innocent.

I advise tonsillectomy for hypertensive patients of any age, if they have evidence of tonsillitis, whether sympathectomy is indicated or not. If sympathectomy is indicated, the patient faces multiple operations. If it is not indicated, perhaps the age is advanced and the general condition is poor. In either case, I believe that local anæsthesia is advisable. I find that my specialist colleagues have become so skilled in this method that the extra burden for the patient is but a minor one.

The Effect of Tonsillitis Upon the Vessel Wall.

Most of the hypertensives whom I have submitted to tonsillectomy have improved in health after the operation, but in only a small minority has there been a significant fall in blood pressure. If the patient is to go through life with a high blood pressure, his well-being and his life may well depend upon the condition of the walls of his blood vessels. It is important therefore to remove any factors which may help to soften or to damage these walls. Vascular complications in the acute infectious diseases, and a study of thrombophlebitis and of *periarteritis nodosa*, provide evidence strongly suggestive of the possible rôle of infection in damaging the walls of blood vessels.

The Present Series.

During my last three years of general practice, I saw 61 patients with hypertension who suffered also from fibrositis and chronic tonsillitis, these representing the great majority of hypertensives who came under my care. Of these 61 patients, 26 had tonsillectomy skilfully performed by various oto-rhino-laryngologists. Of the latter patients 11 have been excluded from study, either because it was not possible to follow them for more than six months, or because they were given dietary instructions which effectively reduced excessive weight. Fifteen patients are thus left for study. For these the tonsillectomy was the only treatment, except for an occasional course of sedatives such as they had already received before operation. Of these 15 patients, 13 noted prolonged and worthwhile symptomatic relief. In four of the fifteen cases a significant and prolonged lowering of the blood pressure was noted.

Aggravation of Chronic Hypertension after Infective Episodes.

Among the four patients whose blood pressure was significantly lowered after tonsillectomy, there is one whose history is worth special attention, firstly because it suggests that infective episodes may aggravate hypertension, secondly because the patient has been under my observation for fifteen years. I first examined him long before I had any idea that there might be a connexion between chronic tonsillitis and chronic hypertension. I give here a brief extract from his clinical history.

Mr. F.H.D. at the first consultation was aged fifty-four years. On October 16, 1933, he said that he had had influenza eight weeks earlier and now felt weak and depressed. His blood pressure was 145 millimetres of mercury (systolic) and 100 millimetres (diastolic). On March 14, 1937, he complained of recent indigestion. One month earlier, when he was in the country, he had developed a sore throat with generalized aches and pains, and had consulted his friend, Dr. Bill Conolly, who said that he had "'flu". His blood pressure was 185 millimetres of mercury (systolic) and 110 millimetres (diastolic). On April 16, 1941, he had had "pains all over" for six or eight weeks. Pus was seen when the tonsils were squeezed. He was given *Mistura Sodii Salicylatis*. On July 7 the pains had gone, except in the right knee. The blood pressure, taken successively in the two arms, was 210 millimetres of mercury (systolic) and 120 millimetres (diastolic) and 235 millimetres (systolic) and 130 millimetres (diastolic). On September 12, 1942, he was giddy, his legs wobbled and he veered to the left. The blood pressure measured successively in the two arms was 210 millimetres of mercury (systolic) and 125 millimetres (diastolic) and 215 millimetres of mercury (systolic) and 130 millimetres (diastolic). On September 24 tonsillectomy was performed, and on October 17 he was back at work. His systolic blood pressure was 195 millimetres of mercury and his diastolic pressure was 115 millimetres. On March 31, 1943, he was well. The systolic blood pressure was 155 millimetres of mercury and the diastolic pressure was 90 millimetres. After operation he dieted so as to lose weight, but his friends told him that he looked haggard, so he allowed his weight to go up again and did not feel any the worse for it. On September 24, 1943, he presented for an annual post-operative examination, and said that he was "feeling wonderful". The systolic blood pressure was 174 millimetres of mercury and the diastolic pressure was 102 millimetres. He requested a certificate for his wife, and took pains to present a case, as if doubtful of the reception of his request. The sphygmomanometer cuff had not been removed from his arm, and the blood pressure rose to 200 millimetres of mercury (systolic) and 115 millimetres (diastolic). The request was readily granted and the subject was changed. The blood pressure fell to 180 millimetres of mercury (systolic) and 100 millimetres (diastolic). On September 1, 1944, he was well; the blood pressure was 190 millimetres of mercury (systolic) and 100 millimetres (diastolic). On February 9, 1945, he was well; his blood pressure was 145 millimetres of mercury (systolic) and 100 millimetres (diastolic). On August 29, 1945, he was well, and said that he felt "a different man since the tonsils were out". His blood pressure taken successively in the two arms was 210 millimetres of mercury (systolic) and 110 millimetres (diastolic) and 205 millimetres (systolic) and 110 millimetres (diastolic). A note was at that time made on his card, questioning whether the rise in blood pressure was due to incipient prostatic obstruction. On April 24, 1946, he was well and had no giddiness. His blood pressure was 200 millimetres of mercury (systolic) and 115 millimetres (diastolic). He had nocturnal frequency of micturition. Soon afterwards a transurethral resection of the prostate was carried out by Dr. Colin Edwards. On October 16, 1947, he was mostly well, and had occasional attacks of dizziness. His blood pressure was 210 millimetres of mercury (systolic) and 115 millimetres (diastolic).

Conclusion.

Whether a throat infection plays any rôle in causing chronic hypertension will no doubt be finally determined by those experimental pathologists who, like Dr. F. B. Byrom, have already thrown so much light upon hypertension. So far, the experimental evidence provides neither proof nor disproof. Dr. Byrom has drawn my

attention to recent work of Trueta and his colleagues. They have produced renal ischæmia by the intravenous injection of staphylococcal toxins. The experiments produced acute lesions, sometimes leading to necrosis of the renal cortex and the death of the animal. So far they have not produced chronic hypertension in this way.

Clinicians should make their contribution by a close study of all possible factors in the individual patient. I am aware that some physicians have submitted to tonsillectomy a few patients with chronic hypertension and they have been disappointed in the results. But disappointment in some cases is to be expected in a disease which is probably due to several different factors, acting through a final common pathway. I am not aware of any substantial and systematic investigation into the question of infected tonsils in chronic hypertension, and until this has been carried out, clinicians should not neglect this or any other factor which may have a probable, or even a possible, relationship to the disease. I would remind you of the words of C. S. Sherrington, that "science nobly declines as proof anything but complete proof; but commonsense, pressed for time, accepts, and acts on acceptance". In the meantime, while he is waiting for the complete proof or disproof, there is an opportunity for the man now starting in general practice or in medicine to begin a study of the data which come to his hand, and to persist with it, until the answer can be given. The task will prove a source of fruitful research and fascinating interest throughout a professional lifetime. For the surgeon, I think that there may be an opportunity sometimes to improve and prolong the good results of sympathectomy by timely tonsillectomy.

Reference.

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AN INVESTIGATION OF THE INORGANIC IODINE CONTENT OF THE SALIVA IN HYPERTHYREOIDISM FOLLOWING THE ADMINISTRATION OF POTASSIUM IODIDE, AND THE EFFECT OF 2-THIOURACIL AND 4-METHYL THIOURACIL THEREON.

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THE capacity of the salivary glands to concentrate iodine has been known for some time; Hammarsten and Mandel (1898) mention the presence of "iodine and bromine combinations" (salts?) in the saliva. Various workers have discussed the appearance of iodine in the saliva after oral, intravenous or parenteral administration of iodides and certain iodine-containing compounds.

Lipschitz (1929) showed that after the intravenous injection of sodium iodide in dogs in quantities of the order of 20 milligrammes per kilogram, there was an immediate maximum concentration of five milligrammes *per centum* of sodium iodide in the blood; there was a 10-fold to 15-fold concentration of inorganic iodine in the gastric juice and a 1·5-fold to 7·0-fold concentration in the parotid secretion; injection of 50 to 100 milligrammes of sodium iodide per kilogram brought about a preliminary high concentration of 15 to 30 milligrammes *per centum* of sodium iodide in the blood, and the concentration of inorganic iodine in gastric juice and saliva was increased accordingly by a corresponding amount.

Barkan and Leistner (1929) showed that when an organic compound designated by them "Jodtropon" (an iodinated albumin) was fed to a dog, organic iodine did not appear in the salivary or gastric secretions.

Lipschitz (1930) showed that after the intravenous injection of large doses of thyroxine in dogs only very small amounts of iodine appeared in the salivary and gastric secretions. He therefore assumed that the small amount of iodine present in these fluids was inorganic iodine, which had been gradually released from the metabolism of the circulating thyroxine.

Schneider and Widmann (1931) estimated the amount of iodine in human saliva; after a resting period they found 10 microgrammes of iodine *per centum*, and after periods of stimulation 76, 0 and 362 microgrammes *per centum*.

Scheffer (1933), whilst carrying out comprehensive studies on iodine balance, found at times considerable excretion of iodine in the saliva.

Heath and Fullerton (1935) examined the rate of appearance of iodine in the saliva (after administration of 0·25 gramme of potassium iodide) in patients with a wide variety of clinical conditions, including achlorhydria or some disease process likely to bring about a disturbance in the mechanism of intestinal absorption. (These workers believed that the delay was a rough measure of the disturbance of the absorptive ability of the upper part of the small intestine; in two cases of myxœdema studied there were lags of 120 minutes and of more than 60 minutes, respectively, before iodine appeared in the saliva.)

Bruger, Hinton and Lough (1941) found that the iodine content of normal saliva varied between only 3·5 and 24·0 microgrammes per 100 millilitres—considerably lower than the figures obtained by Schneider and Widmann (1931).

Flexner, Bruger and Member (1942) reported that after intravenous injection of sodium iodide in man, inorganic iodine was selectively concentrated and secreted by the salivary and gastric glands.

Bruger and Member (1943) noted pronounced increases in the concentration of iodine in the saliva (in man) after oral administration of potassium iodide, of iodine dissolved in poppyseed oil, and of ethyldiiodobrassidate, which they decided first underwent cleavage, because no iodine appeared after the oral administration of sodium tetraiodophenolphthalein. Abnormal amounts of iodine could be detected in the saliva seventy-two hours after ingestion of potassium iodide, and of ethyldiiodobrassidate.

Apparently, therefore, the salivary glands are permeable to inorganic iodine and impermeable to the penetration of iodine in organic combination.

Hence in the present investigations it was decided to ascertain if there was any difference between the amounts of inorganic iodine concentrated and secreted by the saliva in normal and thyreotoxic subjects following the administration of potassium iodide, and also to ascertain, in the case of the thyreotoxic subjects, whether medication (chiefly by 2-thiouracil and methyl thiouracil) had any effect on these values.

Experimental Investigation.

Inorganic iodine in the saliva was estimated as follows:

To one millilitre of saliva, diluted with distilled water (three millilitres) and made acid to methyl orange with 2/3 normal sulphuric acid, was added an excess of fresh bromine water (six to ten drops) and the solution was boiled until excess bromine was removed. After the mixture had been cooled to room temperature a small crystal of potassium iodide was added and the liberated iodine was titrated with N/500 sodium thiosulphate solution (freshly diluted from stock solution), starch being used as indicator (Elmer, 1938).

This method is a rapid one and has some advantage over that employed by Heath and Fullerton (1935), who used ferric chloride as an oxidizing agent in their "spot test", since the saliva contains substances capable of reacting with free iodine and not destroyed by ferric chloride solution (for example, phenol—Diechmann and Schafer, 1942), and probably certain protein apoenzymes containing tyrosine and histidine and also 2-thiouracil and methyl thiouracil, when these drugs were being administered. Because of the fact that Grote's reagent when added to the saliva of patients taking 2-thiouracil gave a persistent green colour (see Grote, 1931), and because of the fact

that 2-thiouracil sometimes causes severe reactions in the salivary glands (as has been shown by Cargill and Lesses, 1945, and by Bishop and Rawson, 1946), the presence of both drugs in the saliva could not be excluded.

A series of investigations was carried out both on controls and on thyreotoxic patients to see whether iodine was normally detectable in the saliva by this method. The saliva was collected at half-hourly intervals over a period of two hours (five samples) and over a corresponding period beginning twenty-four hours later (five samples); in all cases in which iodide was administered, it was given immediately after the first sample of saliva had been collected.

The amount of potassium iodide administered was 0·3 gramme (five grains), equivalent to 229 milligrammes of iodine. The thyreotoxic patients received only one dose before medication was begun under the physician's directions; they received a second dose three weeks afterwards and a third or fourth dose depending on the length of stay in hospital. Employment of a dosage of this size enabled estimations to be made on the inorganic iodine content twenty-four hours after ingestion; furthermore a dosage of this dimension is not regarded as harmful (Marine, 1939, and Thompson *et alii*, 1935). It was also necessary to mask the difference in the thyreoidal and extrathyreoidal iodine requirements between normal and thyreotoxic subjects (Salter, 1940, and Rawson *et alii*, 1945), as otherwise these would have had to be taken into account as a complicating factor difficult of assessment. The iodide was administered in aqueous solution (one fluid ounce) followed by a large glass of water (approximately 250 millilitres).

Eight normal controls (volunteers) took 0·6 gramme of iodide, with a view to finding out whether the amounts of inorganic iodine present in the saliva were doubled when the dosage was doubled.

A further series of investigations was carried out on nine volunteer controls (with leucocyte counts above 6000 per cubic millimetre) who took 0·2 gramme of 2-thiouracil (in gelatin capsules or as tablets) with and without potassium iodide concurrently.

With regard to the normal controls, it was possible in the large majority of cases to perform a series of investigations (of the amounts of inorganic iodine in saliva) extending over periods of six months (in some cases up to twelve months); the averages of the five-hourly figures for the normal controls were themselves averaged and a final figure which could be regarded as characteristic for the individual was obtained; the final figures obtained for the thyreotoxic subjects consisted of the average of five half-hourly values only for the premedication values (however, confirmation was forthcoming in most cases from the values obtained subsequent to medication); in some instances sedation in the form of potassium bromide, chloral hydrate and phenobarbital had been given for several days. This was stopped for twenty-four hours prior to the administration of the iodide.

During periods of medication iodide was given only when the patients were taking thiouracil and no other drugs, or in some instances when administration of the drug had been stopped altogether. In the former circumstances the two-hourly investigational periods were arranged to fall between times when the drug was due to be given; failing this thiouracil was withheld until the five samples were completed.

The normal controls of both sexes were volunteers from the staff of the Kanematsu Institute, the nursing sisters, the lay staff and surgical in-patients (receiving no medication and almost ready for discharge from hospital); hence none of these categories could be described as performing hard manual labour.

The thyreotoxic subjects (of both sexes) provided a mixture of both the primary and secondary (toxic nodular goitre) manifestations of the disease, and also of a type which was specified as diffuse toxic goitre with nodule(s). None of the patients reputedly had a previous immediate history of iodine medication.

Both the controls and the thyreotoxic subjects were permitted a normal routine, except that eating, drinking, smoking, wearing of lip salve *et cetera* were forbidden during the two-hourly investigation periods and for half an hour beforehand; the consumption of "soft" and alcoholic drinks and confectionery was forbidden altogether; the consumption of tea, milk and water was permitted.

As has previously been stated, drugs used for sedation were stopped twenty-four hours prior to the administration of the iodide; however, the effect of adding small amounts of the misturæ singly and in combination to saliva to which known amounts of iodide had been added, was investigated.

Results.

No inorganic iodine was normally detected in the saliva of either the normal controls or the thyreotoxic patients by this method. This may have been due to the fact that the method, whilst being adequate for the investigations under consideration, was insufficiently sensitive to detect the very small amounts of inorganic iodine (0·035 to 0·240 microgramme per millilitre) normally present in the saliva of controls, according to Bruger, Hinton and Lough (1941); the actual sensitivity was estimated to be between 1·0 and 0·5 microgramme per millilitre of saliva; hence it followed that the amounts of inorganic iodine present normally in one millilitre of saliva were too insignificant to affect these observations.

No iodine was detected in the saliva either of normal controls or of thyreotoxic patients immediately after ingestion of 0·3 gramme of potassium iodide solution and 250 millilitres of water, although in all instances, excluding two cases of myxœdema, it was present at the end of the first half-hour. Heath and Fullerton (1935) also reported a considerable lag in the appearance of iodine in the saliva in myxœdema—namely, 120 minutes and more than sixty minutes. These authors reported that the lag period disappeared after medication (with desiccated thyreoid).

There was no outstanding difference between the amounts of inorganic iodine detectable in the saliva of normal controls and in that of thyreotoxic patients during the two hours immediately following ingestion of iodide (the twenty-four hourly figures will be discussed presently).

The actual values showed a range of from 50 to 600 or more microgrammes per millilitre. The majority of "peaks" occurred between the 100 and 500 microgrammes range; in the controls, however, six values exceeded 500 microgrammes per millilitre; of these four exceeded 600 and one of the last-mentioned exceeded 800; in the thyreotoxic subjects five exceeded 500 microgrammes per millilitre, one of these exceeding 600 microgrammes though being less than 700 microgrammes.

The majority of "peaks" occurred in both groups at the one and a half and two hourly intervals, a somewhat smaller number appearing at the hourly interval; only about one-tenth of either population had "peaks" at the half-hourly period.

The eight normal controls who took 0·6 gramme of potassium iodide (10 grains) did not show greatly elevated figures during the two hours immediately after ingestion. The values were somewhat elevated compared with those obtained when 0·3 gramme was ingested, but not doubly so; in the case of the twenty-four hourly figures, however, the values were two to two and a half times as high.

No inorganic iodine was detected in the saliva, either in the immediate period or in the corresponding period twenty-four hours later, when nine volunteer controls took 0·2 gramme of thiouracil only.

The nine normal controls who volunteered to take thiouracil (0·2 gramme) and iodide showed no significant differences between the values obtained for iodide alone both during the immediate and during the corresponding two-hourly period twenty-four hours later.

When small amounts of the sedative drug solutions were added to saliva containing a known amount of

TABLE I.[1]

Normal Controls.			Thyreotoxic Patients.						
Sex.	Age in Years.	x_0 (Micrommes per Millilitre.)	Sex.	Age in Years.	x_T (Micrommes per Millilitre.)	Basal Metabolic Rate *per Centum*.[2]	Serum Cholesterol. (Milligrammes per 100 Millilitres.)[2]	Severity of Symptoms. (*Arbitrary*.)	Diagnosis.[2]
M.	48	21·98	M.	65	138·6	+48		++++	Primary thyreotoxicosis.
F.	26	17·10	M.	37	12·93	+18		++	Toxic nodular goitre.
M.	50	45·02	F.	51	74·72	+42	121	+++	Primary thyreotoxicosis.
M.	27	15·95	F.	37	115·3	+30		+++	Primary thyreotoxicosis.
M.	23	6·52	M.	56	48·34	+34	130	++	Primary thyreotoxicosis.
F.	62	38·32	F.	44	35·94	+16	173	+++	Toxic nodular goitre.
F.	25	5·72	F.	36	67·50	+24	213	++	Primary thyreotoxicosis.
F.	28	18·82	F.	23	23·24	+60	123	+++	Primary thyreotoxicosis.
M.	65	16·70	F.	42	11·62	+46		+++	Toxic nodular goitre.
F.	22	9·31	M.	48	31·66	+26	108	+++	Toxic nodular goitre.
M.	29	8·90	F.	37	41·34	+5	210	++	Primary thyreotoxicosis.
M.	23	12·06	M.	55	34·04	+44	143	+++	Primary thyreotoxicosis.
F.	53	70·10	F.	56	87·70	+35	62	+++	Toxic nodular goitre.
F.	58	35·70	F.	61	38·90	+25	109	+++	Toxic nodular goitre.
M.	43	13·01	F.	26	19·41	+30	95	+	Mild primary thyreotoxicosis.
M.	53	14·17	F.	47	50·86	+25	100	++	Primary thyreotoxicosis.
M.	40	40·40	F.	41	62·34	+22	100	+++	Toxic nodular goitre.
M.	56	70·30	F.	50	121·36	+25	160	+++	Primary thyreotoxicosis.
F.	62	13·29	M.	54	36·82	+28	64	+++	Primary thyreotoxicosis.
F.	57	32·88	F.	31	49·03	+20	43	+++	Primary thyreotoxicosis.
M.	41	22·67	F.	58	98·25	+67	55	++++	Primary thyreotoxicosis.
M.	35	23·44	M.	31	57·90	+36	58	++	Primary thyreotoxicosis.
M.	36	6·23	F.	63	40·80	+29	72	+++	Toxic nodular goitre.
F.	23	11·44	F.	27	47·56	+21	195	++	Primary thyreotoxicosis.
F.	22	20·82	F.	37	21·62	+49	68	++	Primary thyreotoxicosis.
F.	24	22·90	F.	35	31·64	+46	108	+++	Primary thyreotoxicosis.
F.	53	53·63	F.	37	175·96	+37	89	++++	Diffuse toxic goitre with nodule(s).
M.	34	21·54	F.	26	13·40	+47	128	++	Primary thyreotoxicosis.
F.	64	52·22	M.	36	16·18	+47	128	++	Primary thyreotoxicosis.
F.	49	18·46	F.	53	92·06	+35	86	++++	Diffuse toxic goitre with nodule(s).
F.	23	14·42	M.	25	36·54	+26	43	++	Primary thyreotoxicosis.
F.	39	26·42	F.	69	90·66			+++	Diffuse toxic goitre with nodule(s).
M.	57	14·38	F.	30	42·32	+21	92	++	Toxic nodular goitre.
F.	23	77·56	F.	68	62·74	+56	47	++++	Toxic nodular goitre.
F.	40	28·80	F.	31	13·91	+26	71	++	Toxic nodular goitre.
F.	33	4·20	F.	24	56·32	+46	53	+++	Primary thyreotoxicosis.
F.	42	26·09	M.	66	70·76	+42	121	+++	Primary thyreotoxicosis.
M.	38	18·89	F.	43	68·24	+27		+++	Primary thyreotoxicosis.
F.	29	13·79	F.	45	65·68	+62	53	+++	Primary thyreotoxicosis.
M.	28	46·66	F.	61	84·36	+51	200	+++	Primary thyreotoxicosis.
F.	55	37·50							
M.	35	24·74							
M.	62	26·51							
F.	57	51·14							
M.	32	8·14							
F.	68	64·58							

[1] $\bar{X}_0$=27·04; standard error=±2·77. $\bar{X}_T$=57·21; standard error=±5·87. $\bar{X}_{CF}$ (25 females)=30·61; standard error=±4·11. $\bar{X}_{TF}$ (30 females)=60·16; standard error=±6·77.

[2] Data obtained from hospital records.

iodide, in most instances the quantities of iodine found were less than the calculated quantities.

When the figures for the two-hourly periods (twenty-four hours after the ingestion of potassium iodide) were examined for normal controls and thyreotoxic subjects, significant differences were apparent between the amounts of inorganic iodine now present in the saliva. The values for the thyreotoxic patients were those obtained either before any medication had been given at all, or after a small amount of medication, which was stopped twenty-four hours prior to, and during, the administration of iodide. Figure I shows the disposition of the average values for the adult population of normal controls and thyreotoxic subjects between the ages of twenty-two and seventy years; the values for subjects under the age of

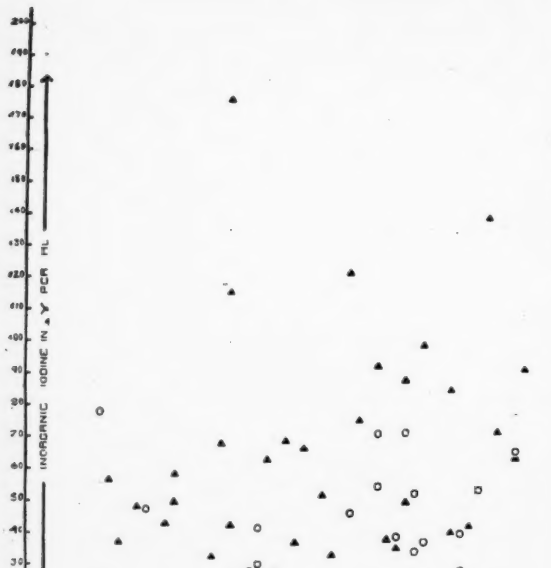

FIGURE I.

twenty-two years have not been shown, because of the fact that only two thyreotoxic patients and ten controls were investigated.

From a general consideration of the disposition of the values shown graphically in Figure I, it would appear that with increasing age there is an increase in the retention of inorganic iodine. In Table I are shown the means of the averages (of five values), namely X_C, for 46 normal controls (of both sexes) and also the X_T values for 40 thyreotoxic patients. The mean $\overline{X}_C$ of X_C values was found to be 27·04 with standard error ±2·77; the mean $\overline{X}_T$ of the X_T values was found to be 57·21 with a standard error of ±5·87; P was found to be less than 0·001, when t was calculated with 84 degrees of freedom. The mean $\overline{X}_{CF}$ of 25 female controls was found to be 30·61 with a standard error ±4·11, and $\overline{X}_{TF}$ of 30 female thyreotoxic patients was found to be 60·16 with a standard error of ±6·77. P was found to be less than 0·001 when t was calculated with 53 degrees of freedom.

The mean $\overline{X}_{CA}$ of ten adolescent controls was found to be 33·35 with a standard error of ±7·01—the values X_{CA} were not included in $\overline{X}_C$ for reasons previously explained.

In Table II have been set out the means X_M for miscellaneous cases investigated. It can be seen that the premedication values obtained for two cases of myxœdema, for one case of non-toxic nodular goitre, and for two cases of anxiety neurosis were in the vicinity of the mean for the normal controls; the values for one case of hypertensive cardiac failure with slightly elevated basal metabolic rate were much higher.

In the two cases of myxœdema investigated there was a lag in the appearance of iodine in the saliva; it was not detectable until the one and a half hourly sample (immediately after ingestion), which is in accordance with the findings of Heath and Fullerton (1935). The lag disappeared when medication in the form of desiccated thyreoid was given, which is also in accordance with the findings of these authors; furthermore, the mean values now obtained for the two-hourly period, twenty-four hours after ingestion of iodine, were very much elevated over the premedication values.

A series of averages obtained for thyreotoxic patients subsequent to medication (with 2-thiouracil and methyl thiouracil) showed variable findings; in some instances the values fell gradually, in others they rose or remained stationary; when the "medication" means were tested against the "premedication" means by the method of paired observations, no significant difference was found.

Discussion

Some of the thyreotoxic subjects were ambulatory and performing occasional light tasks, others were confined to bed—even in bed, however, the thyreotoxic individual is restless. The skin is usually moist and flushed, and considerable water losses occur via this medium; according to Czike and Szel (1935) the total extrarenal excretion of water is four times as much as the renal excretion in hyperthyreoidism, and of this 48% to 58% is lost via the skin; in normal individuals the ratio of the renal to extrarenal excretion was found to be as 1:1; 30% to 40% of the extrarenal excretion was found to be via the skin. However, it has long been recognized that increased perspiration usually occurs at the expense of the excretion of water via the kidneys and *vice versa* (Halliburton, 1891). Further, von Fellenberg (1926) demonstrated that in profuse perspiration the manner of iodine elemination was changed in so far as the excretion via the kidneys was decreased and the elimination via the skin was increased (it would appear that the diminution in the volume of the urine was responsible for this). The actual fluid intake of the thyreotoxic and normal subjects was to all intents and purposes the same, none of the patients being on either an increased or a decreased fluid intake; therefore, it is most unlikely that there was a great difference between the actual fluid losses in the two groups, all media of excretion (lungs, skin and kidneys) being considered, during the comparatively short time of investigation, although the actual mode of iodine elimination and water excretion probably differed in the two cases. Over a longer period differences would become more apparent, as it is well known that the thyreotoxic individual tends towards an ultimate state of dehydration. Furthermore, the activity of the patients was restricted and their food allowance was increased to prevent loss of weight.

As has previously been pointed out, none of the normal controls was engaged in hard manual labour. Now it would appear that the iodine losses via urine and sweat in the normal controls were not of such magnitude as to influence the iodine content of the saliva at the time in question, when a dose of the size employed in this instance was administered, for the following reasons: (*a*) a number of the normal controls who exhibited low iodine values were engaged in comparatively sedentary occupations; (*b*) some of the controls who exhibited high values were engaged in fairly strenuous activities—

TABLE II.
Miscellaneous Patients.

Sex.	Age in Years.	Diagnosis.	$\bar{X}_M$ (Microgrammes per Millilitre.)[1]	Basal Metabolic Rate *per Centum*.	Serum Cholesterol. (Milligrammes per 100 Millilitres.)[2]	Severity of Symptoms. (Arbitrary.)
M.	64	Myxœdema Taking thyreoid (0·5 grain thrice daily) Taking less thyreoid (0·5 grain once only)	20·70[3] 83·02[3] 42·50[3]	−19[3]		++
F.	66	Myxœdema (with controlled *diabetes mellitus*) Taking thyreoid (0·5 grain thrice daily)	17·20[3] 255·8[3]	−16[3]		++
F.	42	Non-toxic nodular goitre	21·02	−12	194	+
F.	23	Anxiety neurosis	22·70			
F.	28	Anxiety neurosis	32·71	−19		
M.	65	Congestive cardiac failure	71·38[3]	+19[3]		+++
F.	56	Hypertensive cardiac failure	18·98[3]	+10[3]	270	++
46 Normal controls (total) $\bar{X}_C$			27·04			
40 Thyreotoxic patients (total) $\bar{X}_T$			57·21			
25 Female controls $\bar{X}_{CF}$			30·61			
30 Female thyreotoxic patients $\bar{X}_{TF}$			60·16			
10 Adolescent controls $\bar{X}_{CA}$			33·36			

[1] $\bar{X}_M$ = Averages for miscellaneous patients.
[2] Premedication values.
[3] Data obtained from hospital records.

namely, nursing sisters; actually the highest value of approximately 70 microgrammes per millilitre was obtained consistently for a control who was engaged in collecting many samples of saliva on time from divers scattered locations; (*c*) in the controls who were confined to bed the iodine values were almost equally distributed between "low" and "high" normal values.

It must next be considered what effect the iodine requirements of the thyreotoxic individual may have on the amount of excess circulating iodine ion. These requirements will not be large, as the involuting effect of iodine on the thyreoid and the detoxication of the patient in Graves's disease have been shown to require only about 6·0 milligrammes of the element daily, according to Thompson *et alii* (1930); hence the amount of iodine administered in this instance (229 milligrammes) should be well able to mask the iodine requirements of the thyreotoxic patient. When these facts are taken into account, it would appear that some explanation other than one based upon iodine excretion and the detoxicating effect of the element in hyperthyreoidism must be found to account for the differences in the retention figures between the two groups.

Since the iodine of the swallowed saliva is reabsorbed by the intestinal tract and finds its way back to the saliva once more via the blood stream, an internal circulation is set up (the "*inner Kreislauf*" of Lipschitz, 1929). Now it might be reasonably supposed from Oswald's (1916) observations that, because of the increased nervous tone and peristalsis of viscera *et cetera* in hyperthyreoidism, the "*inner Kreislauf*" is speeded up in this condition and depressed in hypothyreoidism. Heath and Fullerton (1935) demonstrated the delayed appearance of iodine in the saliva after administration of potassium iodide in myxœdema, and the observations on the two cases reported here confirmed this; the amounts of iodine in the saliva twenty-four hours later were found to be normal. However, it was of interest to observe that the iodine values (for the two-hourly period twenty-four hours after ingestion) were appreciably elevated when desiccated thyreoid was administered; this would suggest that the "*inner Kreislauf*" was being speeded up by the administration of the thyreoid extract. Thus the appearance of the "extra" iodine twenty-four hours later in the saliva in hyperthyreoidism may well be attributed to the metabolic effect of the increased circulating thyreoidal hormone.

Whilst very little is known of the hormonal control of the salivary glands *per se*, nevertheless these glands have been seen to come under hormonal influence in one species; for example, a sexual dimorphism of the submaxillary salivary glands in the mouse has been described by Lacassagne (1940). Hence another explanation is the probable existence of a mechanism to prevent the too rapid loss of exogenous inorganic iodine, and of other substances to which the membranes of the salivary glands are permeable, from the thyreotoxic organism, this mechanism being under hormonal control.

It would be expected that the administration of the antithyreoid drugs would bring the twenty-four hourly figures back to normal limits with improvement in the condition of the patient; however, this occurred only in some instances, so that no significant change could be detected by the method of paired observations. It is possible that the results obtained subsequent to medication were influenced by (*a*) prolonged consumption of certain of the sedative drugs, (*b*) previous iodine administration and (*c*) too short a lapse of time between the premedication and the medication observations.

Summary.

In a series of 40 thyreotoxic and 46 normal subjects investigated, significantly elevated amounts of inorganic iodine (twenty-four hours after the ingestion of potassium iodide) were found in the saliva of the former group.

No significant evidence (by the method of paired observations) could be found to suggest that the administration of the anti-thyreoid drugs 2-thiouracil and 4-methyl 2-thiouracil influenced these findings.

Acknowledgements.

The writer is indebted to Dr. A. J. Canny, formerly Acting Director of the Kanematsu Institute, Sydney, who suggested that the investigation should be undertaken; to Miss Ailsa McInness for her willing assistance; to those members of the staff of the Institute, the nursing sisters and the lay staff who acted as volunteers; to Dr. R. M. Allen, Dr. L. Dunlop, Dr. W. Evans, Dr. A. W. Holmes à Court, Dr. E. H. Stokes and Dr. G. Willcocks for permission to investigate their cases;

to Miss Lorna Gibson (librarian of the Sydney Hospital Central Library); and to Professor V. M. Trikojus (University of Melbourne) for some criticism of the paper.

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A SIMPLE ABDOMINAL RETRACTOR.

By Keith J. B. Davis,
Sydney.

The new model of a retractor for abdominal surgery described in this paper is presented because of its simplicity and of the absence of superstructure, and for the reason that when the blades are placed in position they will not slip.

There are two upper abdominal retractors in common use in Australia, the Devine and the Humphrey. Both of these have done yeoman service, but they both have certain defects inherent in their construction. The Devine retractor is the more elaborate of the two; but both have much superstructure which projects above the level of the basic frame. The disadvantage of this is that ligatures and suture material, sponges and gloves are apt to be caught or twisted round the various hooks or wing nuts of the retractor. There are also a number of removable parts, which may become unscrewed and are apt to find their way into the abdomen. The method of anchoring the blades of the retractor is not entirely satisfactory, as they may slip and have again to be placed in position. In the Humphrey retractor, however tightly the wing nuts may be screwed, it is found that during a prolonged operation the blades of the retractor slip, especially if pressed upon from the periphery.

The present retractor is simplified, in that there are only twelve small studs no higher than three-eighths of an

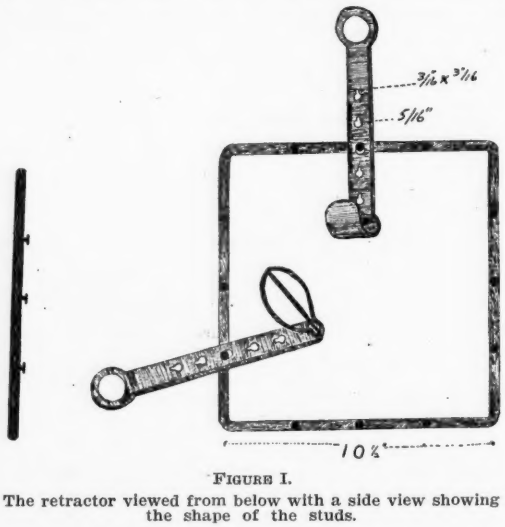

Figure I.

The retractor viewed from below with a side view showing the shape of the studs.

inch, all of which are incorporated in the retractor, and although these are fixed in position, it is thought that they give sufficient scope for placement of extra retractors in addition to the four fixed anchoring blades. The corners are rounded. The frame is ten and a half inches square, and this area, in view of the manner with which the blade handles are slotted, provides sufficient room for opening any abdominal wound. The frame is three-eighths of an inch wide and thick.

The blades of the retractor are of the standard pattern, the shanks being slotted with a circle five-sixteenths of an inch in diameter and extended to an oval three-sixteenths of an inch by three-sixteenths of an inch; five are placed in each blade one inch from each other. The shank is ended in a circle, which serves for traction and holding. The four anchoring retractors are placed in position in the wound, and the frame is then superimposed on them with the studs lying towards the abdomen. Each retractor is then slipped over the stud and the shank of the stud falls into the small oval hollow of the slot. Each retractor is treated in this manner until all four are in place. Should the "hand" be required, the shank of this retractor is slid under the frame, the "hand" is placed in the required position, and the appropriate slot is anchored onto the stud in the manner indicated above. The removal or adjustment of any blade is simple, and the shank of the blade can easily be freed from its stud. Thus the retractors are completely fixed and can move only if the surgeon alters the position deliberately; even pressure on the handle at the end of the blade will not alter its

position. The tighter the muscles of the abdomen become, the more fixed the blade.

Acknowledgements.

In the manufacture of this retractor I have to thank Mr. E. Wetherill, of C. H. Mitchell and Company, Limited, Oxford Street, Woollahra, who has offered valuable help and suggestions. I also wish to thank Miss P. Davis who executed the diagram. The retractor is not at present purchasable at any of the surgical houses.

Summary.

1. A new and simplified model of an upper abdominal retractor is presented.

2. This retractor has no superstructure above the frame, and its blades, when once fixed, will not slip.

Reviews.

MALARIA.

DR. W. K. BLACKIE is consulting physician to the Salisbury Hospital in Southern Rhodesia. In this monograph, "Malaria with Special Reference to the African Forms", he gives an account of the parasitology, pathology, diagnosis and treatment of that disease.[1]

The chapter headings give an idea of the scope of the book. They are: 1. "Historical Review." 2. "Parasitology." 3. "Pathology and Morbid Anatomy." 4. "Clinical Features." 5. "Diagnosis." 6. "Differential Diagnosis." 7. "Prognosis and Treatment." 8. "Prevention." The monograph has been designed for the use of the physician, and the chapter on prevention is concerned only with chemoprophylaxis.

Most of the errors which are copied from textbook to textbook of tropical medicine have been avoided, but there are a few points that deserve to be mentioned. It is unfortunate that the single coloured plate of the species of plasmodia, which is reproduced from the "British Encyclopædia of Medical Practice", should include *Plasmodium tenue*, which is not mentioned elsewhere in the book. Dr. Blackie accepts the evidence obtained from the use of therapeutic malaria in neurosyphilis as indicating that natural immunity to malaria, although rare, does occur. This question is almost impossible to decide, but it is fallacious to place much weight on data derived from patients of this type, as premunity, attenuation of the parasite, and even arsenic therapy may confuse the picture. In over a thousand healthy non-immune volunteers infected at Cairns with either *Plasmodium falciparum*, *Plasmodium vivax* or *Plasmodium malariæ*, Fairley and his colleagues did not encounter one case of natural immunity.

It has long been held that the concurrent administration of "Plasmoquine" and "Atebrin" accentuates the abdominal pain which sometimes occurs during "Plasmoquine" therapy. Dr. Blackie agrees with this. Australian army experience with therapeutic trials involving over a thousand men failed to support this view, the incidence of complications of "Plasmoquine" therapy being the same whether "Atebrin" was stopped two days before the "Plasmoquine" was begun or whether the two drugs were given concurrently.

There is no mention, when the use of "Atebrin" as a suppressive is discussed, of the severe dermatitis ("atypical *lichen planus*") which was found to be an occasional complication of the continued use of that drug, or of the very rare occurrence of aplastic anæmia.

Laboratory workers in England succeeded recently in establishing strains of bird malaria parasites which were resistant to large doses of "Paludrine". Dr. Blackie discusses the therapeutic and prophylactic use of "Paludrine" in considerable detail, and in an addendum he remarks that some Rhodesian strains of *Plasmodium falciparum* fail to respond rapidly to therapeutic doses of that drug, and also that severe attacks of malignant malaria have occurred in spite of systematic "Paludrine" prophylaxis. This experience, which resembles that of the "Atebrin"-resistant strain of *Plasmodium falciparum* found at Aitape and Wewak in New Guinea, emphasizes the need for careful local evaluation of the effectiveness of all new drugs.

The description given of acute and chronic malaria in the African would apply equally well to the natives of malarious areas of New Guinea and adjacent islands, although certain of the diseases mentioned in the differential diagnosis, namely, schistosomiasis and kala-azar, are of no importance in the Australasian region.

There is a good index, but no references are given.

The monograph can be recommended to any physician likely to be concerned with the diagnosis and treatment of malaria as a readable, well-balanced, practical and up-to-date account of the subject.

THE APPENDIX.

THE appearance of "The Appendix", by R. J. McNeill Love, makes anyone surgically inclined curious to scan its contents.[1] One finds a compact little book, clearly illustrated, and written in the author's usual concise style. The whole problem of appendicitis is thoroughly covered and answered. Sherren's influence is acknowledged and the case for the delayed treatment of appendicitis is clearly stated.

One learns of a growing tendency to omit ligation of the appendix and to remain content with the approximation of its walls by crushing and the purse-string. Drainage through a grid-iron incision is rightly condemned. One notable omission is the absence in the treatment of post-operative thrombosis of any reference to anticoagulants or venous ligation.

Of passing interest to senior men, this comprehensive book is an invaluable guide to students and younger practitioners.

OPHTHALMOLOGY IN CHILDREN.

"DISEASES OF CHILDREN'S EYES", by J. H. Doggart, is described by the author in his preface as a volume devoted exclusively to children's eye troubles, written in the hope of arousing wider interest in the ophthalmic problems of childhood.[2] Whilst all ophthalmic surgeons could profit by studying it, this book will be of value particularly to those ophthalmic surgeons who have never worked in children's hospitals and to those on the threshhold of their career.

From his association with the Children's Hospital, Great Ormond Street, London, the author is well qualified by experience to deal with his subject. The book also reveals ample evidence that the author possesses the knowledge of the right approach to and sympathetic management of the young patient and the immediate relatives.

The book itself is comprehensive. The first chapter gives an excellent outline of the principles of examination. Then follow four chapters on the anatomy of the eye and its adnexa. The development of the eye and its adnexa and the congenital abnormalities relating to these structures are described in four chapters. Ocular complications and sequelæ of systemic diseases, methods of treatment, ocular welfare, injuries of the eyeball, and disease of the orbit are dealt with in that order.

The chapter on refraction is particularly good, though it is regretted that the word shadow is retained in the discussion on retinoscopy. One does not appreciate a statement such as "the shadow will increase in speed and brilliance".

Disorders of movement (incomitant and comitant squint, heterophoria, nystagmus *et cetera*) occupy five chapters; it is here particularly that much helpful advice is given on the true appreciation of the child's problem and the correct approach to its solution. This section is one which should be helpful to the "children's physician and family practitioners" for whom the author is in part writing. In this connexion, however, it is regretted that in the treatment of paralytic squint no mention whatever is made of occlusion of one eye to obviate diplopia. General practitioners unfortunately so consistently fail to take just this measure, simple yet essential, that insistence on it would seem to be of prime importance.

In dealing with what he describes as the "true" or "paretic" alternator type of congenital squint, the author

[1] "Malaria with Special Reference to the African Forms", by W. K. Blackie, M.D., Ph.D., F.R.C.P. (Edinburgh), D.T.M. and H.; 1947. Cape Town: The African Bookman. 8¼" × 5½", pp. 112, with illustrations. Price: 10s. 6d.

[1] "The Appendix", by R. J. McNeill Love, M.S. (London), F.R.C.S. (England), F.A.C.S.; 1947. London: H. K. Lewis and Company, Limited. 7¼" × 4¾", pp. 202, with many illustrations. Price: 12s. 6d.

[2] "Diseases of Children's Eyes", by James Hamilton Doggart, M.A., M.D. (Cantab.), F.R.C.S. (England); 1947. London: Henry Kimpton. 9¼" × 6", pp. 300, with many illustrations, some of them coloured. Price: 42s.

states that ". . . the parents usually implore us to operate early . . ." and "there is no good reason why we should not operate on such cases in the fourth year of life". Many surgeons would consider that this was unduly late to operate in this type of case.

One chapter is devoted to diseases of the lachrymal apparatus, and the rest of the book deals with diseases of the various structures of the eye. The commoner diseases are described briefly yet sufficiently, and most of the four diseases receive mention.

In a volume published in 1947 one expected to find some reference to retrolental fibroplasia. One cannot agree with the author's statement that in spring catarrh the "cobblestones" are found closely set in the lower palpebral conjunction and that this incidence in the lower lid is of help in the differential diagnosis from trachoma. Experience in this country is that the cobblestones are always in the conjunctiva of the upper lid, the lower conjunctiva being smooth but showing the watered milk appearance.

The book is very freely illustrated, well indexed, and the general production is of a high standard. The author's style is fluent, making the book very easy and enjoyable to read.

Many of the opinions expressed in the book are the result of the author's personal experience; any criticisms of minor details would be similarly mere personal opinions. There can be no criticism of the author's general teaching and principles. This is a book which any ophthalmologist could read with profit to himself and benefit to his patients.

PROGRESS IN ENDOCRINOLOGY.

IN the sixth edition of "Recent Advances in Endocrinology" A. T. Cameron, Professor of Biochemistry in the University of Manitoba, gives a very complete review of modern views of the subject.[1]

The interest in thyreoid disease which has been quickened by the introduction of anti-thyreoid drugs is reflected in a very thorough presentation of the progress in the study of the functions of this gland.

The author mentions synthetic iodoproteins with marked thyreoid activity which have been prepared by allowing simple proteins, for example, casein, to take up iodine under suitable conditions. Hydrolysis of resulting compounds yields thyroxine and diiodotyrosine with ease. Further studies of the optimum conditions of chemical reaction have resulted in iodoproteins of relatively high thyreoid activity, and the commercial possibilities are obvious. Hyperthyreoidism and its modern treatment are adequately dealt with.

The progress in treatment of Addison's disease with subcutaneous implants of pellets of synthetic desoxycorticosterone acetate ("D.O.C.A.") is recorded in the case of a physician who had maintained normal health over a period of six years by this means. Pigmentation disappeared and patient comments "it would not be possible to diagnose the disease clinically".

Altogether this edition is an excellent summary of our present knowledge of an expanding branch of medicine. Each section has a full bibliography. This book is one of the best reviews of endocrinology and its clinical implications, written for the average medical reader, that has come under our notice.

INTERNAL MEDICINE.

MCCOMBS'S second edition of "Internal Medicine in General Practice" contains very up-to-date information on all branches of medicine.[2] The book is in no sense a synopsis, but the descriptions are clear and concise. It is easy to study any subjects as there is no waste of words or padding.

The hæmopoietic system is especially clearly discussed and a study of this section would be useful to any practitioner. Routine investigations are also adequately described.

There has been no attempt to discuss the many classifications of nephritis, a procedure which only makes a difficult subject more clouded with doubt. The method chosen is clear and appears to fit the case well. The various sections on gastro-enterology and cardiac and chest diseases contain all relevant information.

The sulphonamides, penicillin and streptomycin are fully described. The dangers of the sulphonamides are strongly stressed and there are few practitioners who have not seen tragedies due to this drug. This section should help to lessen the free use of the sulphonamides in any pyrexia or simple disease. Criticism may be levelled at the inadequate mention of the risks associated with the topical application of this drug.

Allergy is well discussed and the illustrations are useful.

The part played by nervous factors appears in the opening chapters to be overstressed, but the later pages reveal a well-integrated outlook on this aspect. An important feature is stressed, namely, that neurosis should be a positive rather than a negative diagnosis.

Illustrations are particularly clear and modern, and diagrams serve a useful purpose.

This book contains a mine of information in a well-compressed form and should be useful for quick reference on any medical problem.

A TEXTBOOK OF SURGERY.

THE fourth edition of Professor C. F. W. Illingworth's "A Short Textbook of Surgery" is an excellent textbook for students.[1] Space has not been wasted by the description and illustration of operations, the technical details of which are not the concern of the undergraduate. The book begins with a section on safety factors in surgery, the author stressing a careful history and physical examination. Each regional section begins with a detailed description of the technique of clinical examination for that particular part. In this way emphasis is laid on methods which are all too often neglected in favour of laboratory reports. A section on "bone-setting" is included, and the book ends with a section on rehabilitation, which can be taken as examples of the sound practical value of the book. The more difficult words are made clear by an indication of their Greek roots.

A little criticism cannot be avoided. Streptomycin and "Dicumarol" are not described. The Rh factor is discussed, but there is no mention of the great importance of the avoidance of Rh-positive blood in the treatment of Rh-negative women in the child-bearing age. For cerebral angiography, "Diodrast" might well replace the radio-active "Thorotrast". As so often happens, the layers of the hydatid cyst have been misnamed, the adventitia being called the ectocyst. Recent advances in the surgery of lung abscess have not been incorporated, and pulmonary surgeons would probably object to the term pulmonary "collapse" (instead of atelectasis) and above all to its description as "not dangerous". The advice to place two drains in the peritoneal cavity when the appendix has ruptured would be deplored by surgeons who have advanced beyond this old "period" practice. Even the incision for high ligation of the saphenous vein is drained. And the common duct is drained externally after choledochotomy! The spirit of Alexander MacCormick might well sigh and remember the proverbial neglect of prophets in their own country.

In acute peritonitis the surgeon is advised to keep the bowels at rest for a week or more, but to give turpentine enemas for distension (page 488). And, of course, distension occurs in all cases. In the non-operative treatment of acute appendicitis "the first aim is to keep the inflamed parts absolutely at rest" . . . "but the bowel may be opened by enemas" (page 586). "Emergency cases, operated upon without preparation, are no more liable to complications than others." As a routine, therefore, ". . . an aperient and soap and water enema are given before operation" (page 2). These are repeated after operation (page 4). And so we need not be surprised that "gas distension of the abdomen is so common a post-operative complication as almost to be regarded as the rule" (page 9). Here we have a delightful example of that contradiction and confusion which so often nonpluss the student and which have recently been discussed and explained in this journal.

But the faults are few and there is a great deal that is good.

[1] "Recent Advances in Endocrinology", by A. T. Cameron, C.M.G., M.A., D.Sc. (Edinburgh), F.R.I.C., F.R.S.C.; Sixth Edition; 1947. London: J. and A. Churchill, Limited. 8" x 5¼", pp. 452, with many illustrations. Price: 21s.

[2] "Internal Medicine in General Practice", by Robert Pratt McCombs, B.S., M.D., F.A.C.P.; Second Edition; 1947. Philadelphia and London W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¼" x 6¼", pp. 758, with many illustrations, some of them coloured. Price: 56s.

[1] "A Short Textbook of Surgery", by C. F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S. (Edinburgh); Fourth Edition; 1947. London: J. and A. Churchill, Limited. 9¼" x 6", pp. 688, with many illustrations. Price: 30s.

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THE ASSOCIATION AND THE PERTH CONGRESS.

MEMBERS of the British Medical Association, wherever they may find themselves, are bound to one another by the objects of the Association—they are pledged in the first place to further, by research, by study and by practice, the medical and allied sciences, and secondly to maintain the honour and interests of the medical profession. The second objects which are bound up with professional conduct in relation to brother practitioners, the patient and the State—with etiquette and ethics—need not concern us at the moment. The furtherance of the medical and allied sciences is the real reason for the existence of the British Medical Association, and this cannot be over-emphasized. The medical practitioner is charged with the care of the community in the prevention and cure of disease. This is a trite saying, but it is none the less worthy of repetition in the present instance. To forget it, even for a short space of time, is to take a first step on the road that leads to deterioration. Three methods by which the medical and allied sciences may be promoted have been mentioned—research, study and practice. These are probably realized and understood by every reader of this journal, and many could no doubt write an effective essay on the proper attitude of medical practitioners to them. Such a task will not be attempted at present. The point that should be emphasized is that for the medical practitioner research, study and practice are successive steps of progress towards his objective. The promotion of medical and other sciences does not rest solely with the research worker, of basal importance though his work undoubtedly is. The investigator in the laboratory makes a discovery and he with others, or maybe alone, sometimes finds a practical application for what he has found. Possibly the clinician finds the application for what the research worker has laid bare. We know, of course, that the clinician may be the one to make a discovery. No better example of this could be found than the observations of N. McAlister Gregg, of Sydney, in regard to the occurrence of congenital anomalies in the eyes of children whose mothers suffered from rubella in the early months of pregnancy. New work is reported in medical and other scientific journals, and opportunity is given for study by a wide circle of readers; further, we all know how experience in everyday practice may modify original conceptions of treatment by new methods. But new knowledge is not spread and applied only by the printed word. At the risk of having to face an accusation for stressing the obvious we must once again declare that knowledge spread by word of mouth is much more effective than anything appearing in books or journals, especially if opportunity is given for questions and arguments about it. This is why the several Branches of the British Medical Association hold scientific meetings at stated intervals and try to bring their members together. It is also the reason why in the larger sphere of the Commonwealth as a whole the sessions of the Australasian Medical Congress are held and practitioners from all parts of Australia are given chances of meeting that they would not otherwise enjoy. Anyone who sets out to study the medical history of Australia will soon discover that from the time when the first Australasian Medical Congress was held at Adelaide in the year 1887, the reports of successive sessions are a faithful record of the progress of scientific medicine in this country. Twice there has been a forced break in the continuity of congresses by war—the first occurred because of the first world war, before the congress was controlled by the Federal Council of the British Medical Association in Australia; the second took place after the fifth session of the Association's congresses had been held in Adelaide in 1937. This brings us to the sixth session, which will be held at Perth from August 15 to 21 of this year, under the presidency of Dr. D. M. McWhae.

The Perth Congress has been mentioned in these columns on several occasions and members of the Branches have been urged to attend. This is not the last occasion on which the same plea will be made. To confer on subjects of medical interest is good; to confer in pleasant surroundings is better. In another place in this issue will be found an article descriptive of Perth and its beauties from the pen of Professor Walter Murdoch, whose eminence as a writer is acknowledged by all intelligent people in the community. Those who have not visited Perth will find that it has qualities and attractions peculiarly its own and that the people have that open-handed hospitality which is characteristic of those with generous minds. Practitioners who have been to Perth will, in regard to venue, need no persuasion to become members of congress. The architecture of the University of Western Australia and its unrivalled situation on the banks of the River Swan combine to make an ideal setting for a congress. As far as can be judged at present, subjects of interest covering a wide range will be dealt with at the meetings of the sections. In several centres of medical activity original observations are being collated and will be presented. Several of the special groups within the Association have arranged to combine their gathering with sectional meetings of congress or to meet in Perth close to the date of congress. Among these are the Ophthalmological Society of Australia (British Medical Association) and the Australian Society of Anæsthetists. As already announced, the plenary session will be devoted to the subject of pulmonary tuberculosis and the speakers selected are all masters of their aspects of the tuberculosis problem. At this stage the suggestion may be made that a meeting of congress is a most suitable occasion for the

presentation of papers dealing with medical problems that affect the whole of Australia, particularly in regard to the life and nutrition of the people and the prevention of disease. At the same time it must be clearly understood—and the statement is made chiefly for the general public and not for the members of the medical profession, most of whom know it—that medico-political matters can have no place in congress discussions.

It has been well said that nothing succeeds like success, and we are in the fortunate position of being able to state that already many members of the Branches have asked for membership. Accommodation in Perth will be severely taxed and it is desirable that applications should be sent to the honorary (local) secretaries without delay. The names of the honorary (local) secretaries are set out in "Congress Notes" on another page in this issue. If a decision to apply is left too long there is risk of disappointment. Some months ago, in reply to a statement that it was a long way from Melbourne to Perth, Dr. F. W. Carter, one of Western Australia's representatives on the Federal Council, remarked that it was no further from Melbourne to Perth than it was from Perth to Melbourne. Representatives from the Western Australian Branch make the journey to Melbourne and Sydney with unfailing regularity. They have every right to urge the members of all the other Branches not to forsake "the assembling of ourselves together", but to "hold fast the profession of our faith without wavering".

Current Comment.

ILLUMINATION.

THE increasing popularity of modern methods of lighting indicates a growing awareness that satisfactory illumination is important. However, there is still a great deal of unsatisfactory artificial illumination of homes and workplaces, and very little general knowledge of the principles involved. In opening a discussion on the subject at a meeting of the section of ophthalmology of the Royal Society of Medicine,[1] J. G. Drummond Currie suggested that ophthalmologists, in addition to providing a prescription for spectacles, should go and see for themselves the conditions under which the spectacles are used, not confining themselves to establishments of modern scientific design and lighting, but also visiting those less favoured. Currie then discussed the factors concerned in the provision of satisfactory lighting, lack of which causes strain and decreased efficiency, and described the three main sources of light in general use: the metal filament lamp, which is cheap in first cost, convenient and, if rightly used, fairly efficient, although it has disadvantages in regard to heat dissipation and colour matching; the mercury vapour or sodium lamp which is more expensive to instal and cheaper to run, but lessens colour values; the fluorescent tube which is generally satisfactory. Two objections have been made to fluorescent lamps: stroboscopic defects which are only sometimes apparent and can frequently be minimized; and fear that the lamps may emit harmful ultra-violet rays, a fear which Currie (and subsequent speakers) considered groundless. He concluded with five rules in the provision of lighting. Firstly, light should be adequate for the type of work and from a suitable source as regards lamp and fitting. Secondly, the source of light should not be too near the direct line of vision. Thirdly, shadows should be avoided as much as possible. Fourthly, specular reflection and too much heat are disadvantages. Lastly, too great a contrast of surrounding objects can be tiring and even dangerous.

The measurement of illumination and brightness was considered in some technical detail by J. W. T. Walsh, of the National Physical Laboratory. He pointed out that the assessment of brightness in terms of the photometer was not sufficient; there was also a "subjective brightness", and the two should not be confused.

H. C. Weston, of the Medical Research Council, in discussing the illumination requirements of the eye, referred to the often expressed idea that artificial light is not good for the eyes. His view is that, although artificial light cannot be made identical with daylight, it is usually "white" light, and it has "never been shown that healthy eyes suffer any harmful effects which can be attributed to the 'make-up' of the luminous flux emitted by such sources", that is, sources of "white" artificial light. In his experience most of the headaches blamed on to fluorescent lighting have not appeared to be ocular in origin. Badly planned illumination will, of course, be a source of dissatisfaction with all types of light source. Artificial light can be bad for the eyes not because of qualitative dissimilarity to daylight, but because there is not enough of it. The assessment of the amount of illumination needed for particular tasks can be made in several ways. One can ascertain the illumination currently used for different purposes; but this is unreliable, as economy often makes a degree of lighting customary which would be greatly exceeded if those using the light had free choice. Experimental determination may be made of the level of illumination at which the performance of particular visual tasks comes to a maximum; this determination has been made for some specific occupations and the findings should be capable of application to others. A third method is an elaborate analysis of the specific task and the objects involved in the light of certain basic knowledge; for practical application this method has been simplified by the preparation of a chart which is included in "The Illuminating Engineering Society's Code for the Lighting of Building Interiors". As illustrations of the widely varying requirements, a degree of illumination of seven lumens per square foot is recommended on cookers, sinks and tables, where there are specific visual tasks to be done, 30 lumens per square foot for the weaving of medium worsted fabrics, and 300 lumens per square foot on operating tables. A further aspect brought out by Weston is that there should be no great difference between the brightness of a restricted field of interest and that of the surrounding general field of view. This is necessary for the development of a high degree of acuity, to avoid constant readaptation as the gaze is shifted, to avoid an impression of gloom and to reduce glare. Weston went on to emphasize the importance of glare due to unsuitable or badly arranged sources of light or to reflections from shiny surfaces; a complaint that light is "too bright" often means only that the sources of light are glaring. If glare is avoided high degrees of illumination are unlikely to be harmful. Finally Weston referred to the decline in visual acuity with advancing age, with loss of transparency of the media, particularly to blue light, and reduction in the size of the pupil; this is quite apart from the depreciation of accommodation known as presbyopia. Its occurrence is insidious and may be related to changes in the central nervous system, but the important thing is that increased illumination is a necessity, even in many cases for those under the age of forty years, and can largely offset the decline in visual performance.

In the subsequent discussion the matter of glare received a good deal of attention. The use of glazed paper in books was questioned and it was explained that the contrast range between good white paper and black ink was high (30 to 1); this is necessary for bad lighting conditions, but is too great for a well-lit room. Brian Stanford remarked that most homes were ill-lit, but books designed for the richer people who had brightly lit homes were printed on off-white paper, so bringing the contrast down to about 20 to 1. The same speaker made an interesting comment on the alleged "strain" on the eyes

[1] *Proceedings of the Royal Society of Medicine*, November, 1947.

due to fluorescent lighting. His suggestion was that artificial light, while mostly much less than daylight, was used to supplement waning daylight. The modern fluorescent "daylight bar" produced an effect so like daylight in quality, though only equivalent in quantity to other artificial lighting, that one had a sensation of wanting to switch on the artificial light, although it was already on! The substitution for the "daylight bar" of a bar that gave a reddish light produced an effect of artificial light and, though the level of illumination was much the same, the result was more acceptable. Perhaps this touches closely on one of the essential aspects of the whole problem of illumination—the psychological effect. No scientific measurement or arrangement will be satisfactory if the individual is not subjectively satisfied. This is probably at the heart of the problem of tinted goggles about which there was some discussion. They are very popular, though the scientific indications for their use are quite limited. They appear to answer some subjective, if not an objective, need. The president of the section, Harold Levy, thought it might be said that they were worn often merely as a contrast, just as the black patch was worn in earlier days.

THE PREVENTION OF MENTAL DEFICIENCY OF PRENATAL ORIGIN.

THE term "mental deficiency" can perhaps be stretched somewhat to include not only those conditions in which the exercise of the higher functions of mind is limited by damage or aplasia of the brain, but also serious defects such as those leading to blindness and deaf-mutism. A great deal of information has accumulated during recent years concerning pre-natal causes of these lamentable handicaps; in fact, the knowledge is so recent that its implications have hardly yet been realized. This is pointed out by Peter Gruenwald, who has briefly reviewed what we know of the pathological causes of subnormal mental development.[1] Many special fields are traversed, but there is one common ground, that is, the hope of lessening the numbers of misfortunes affecting the hapless children concerned, their families, and the community in which they live. Gruenwald quotes Benda, who estimated that 2% to 4% of the population have some degree of subnormal mental development, and further that from 30% to 40% of severe mental defects arise in families who could expect normal offspring. Of course there are many hereditary traits which can produce mental deficiency, but without entering the field of eugenics it is valuable to think over the wide variety of prenatal causes. The author has drawn up a table setting out the known causes. After hereditary defects he naturally places syphilis, about which we may perhaps feel a greater optimism, except that the increased incidence of the infection and the recent evidences of a less sensitive community conscience in such matters are disquieting. Toxoplasmosis is perhaps rare, but the recent publication of a case in this journal may have made Australian readers wonder if it has been overlooked in the past. Rubella, of course, is still "news" in this sense, and the importance of virus infections during the early months of pregnancy is well understood. Endocrine deficiencies in the fœtus are next on the list, and here the author includes cretinism and, under the heading of pituitary defects, mongolism. He admits, however, that in spite of much study the latter subject is not settled. There are some points in favour of genetic factors, but the pathological changes support the pituitary theory in general. As the changes in the mongol child must occur at a very early stage in fœtal life, it is possible that various factors may contribute to the curiously widespread and characteristic defects in structure and function. Blood group incompatibilities, besides causing erythroblastotic anæmia, are known to produce kernicterus in the severer forms affecting the nervous system, but in addition the author quotes recent surveys which indicate that milder types of mental deficiencies may also be due to the same cause. The pathological mechanism is not clear, but the statistical evidence is suggestive. In addition to all these possible causes of mental defects in the newborn there are the more purely mechanical ones, due to birth stresses of various kinds, which in turn give rise to direct damage or anoxia, with its possible baleful effects on high-grade cellular structures.

Mental deficiency, blindness and deaf-mutism now interest a great diversity of practising doctors, researchers and social workers, and, as Gruenwald states, we can no longer say which of their causes are beyond our control. We do not know what proportion of the pathological states included in these categories is due to any particular causes or groups of causes, but it is important that we should know. Circumstances of life are now forcing thoughtful people more than ever to plan their families; it is not necessary to enter into controversy to recognize this today. Nor is it necessary to labour the point of the value of quality. Gruenwald's concluding sentence may be quoted: "The cost of the most extravagant research program in this field may well turn out to be lower than the savings of our communities resulting from a reduced number of inmates in their institutions."

[1] *The American Journal of the Medical Sciences*, December, 1947.

MIGRAINE.

THE diagnosis of pains in the head is beset with difficulties, and attempts to discover accurate methods for differentiating one type of head pain from another are to be encouraged. However, it must be admitted that there is at times a lack of agreement among the experts. A clinical test for the diagnosis of migraine has been described by M. T. Schnitker and M. A. Schnitker,[1] which they claim to have found specific. After a careful neurological examination to rule out intracranial pathological conditions, the patient is given one-fiftieth of a grain of nitroglycerin placed under the tongue; within five or ten minutes, according to these investigators, in migraine subjects a typical migraine headache comes on; syncope may occur but is rare. "After the patient has thus been given a headache", as the report so nicely puts it, "it is only right that it be relieved"; this is done by compressing the carotid artery on the involved side for one or two minutes, by giving the patient pure oxygen to breathe for five minutes, or by injecting 0·5 to 1·0 milligramme of ergotamine tartrate intravenously. Without treatment, it is stated, this headache will subside in ten or fifteen minutes. If nitroglycerin does not reproduce the type of headache usually complained of, after an interval of fifteen minutes histamine base is injected subcutaneously according to the method previously described by B. T. Horton.[2] If a typical headache develops, this is designated histamine cephalalgia.

Underlying these tests is the fairly well-supported theory that migraine is due to the stretching of the cranial arteries, chiefly the extracranial structures (the external carotid artery and its branches), and that, conversely, headache associated with histamine or fever is due chiefly to dilatation of cerebral branches of the internal carotid, vertebral and basilar arteries.

If migraine is diagnosed by this test, these authors give the patient ten grains of calcium lactate twice a day together with a sedative such as one-half grain of phenobarbital twice a day, and eliminate as far as possible substances which produce an allergic response, particularly coffee, chocolate, beans, peanuts and "Coca-Cola". At the onset of an acute attack they administer two to four milligrammes of ergotamine tartrate placed under the tongue. By these methods M. T. and M. A. Schnitker claim that they achieve good results. The average practitioner may, however, find it difficult to classify his results quite so easily, and he may find response to treatment by the régime mentioned somewhat unreliable. Some confusion still exists in classifying migraine and histamine headaches, and treatment is often unsatisfactory.

[1] *The Journal of the American Medical Association*, September 13, 1947.

[2] *The Journal of the American Medical Association*, February 1, 1941.

Abstracts from Medical Literature.

PÆDIATRICS.

Human Milk Bank in a Maternity Hospital.

JOYCE WRIGHT AND ELSIE M. C. EDWARDS (*The Lancet*, August 16, 1947) describe the establishmet of a frozen human milk bank in a maternity ward and an experimental milk bank in a laboratory. Human milk was obtained from the mothers in one twenty-bed ward. It was collected by a clean ward technique, pooled, boiled and stored in sterilized bottles sealed by a wool plug and "Cellophane" cap in an ice-cream freezer at −12° C. The milk was successfully used for feeding premature babies and infants with gastro-enteritis, pyloric stenosis and nutritional disorders. The milk in the experimental laboratory bank was stored in the same way. The sterility and flavour of the milk in this bank were tested immediately after boiling and at the fourth, eighth and twelfth weeks of storage. There was a very slight deterioration of flavour between the eighth and twelfth weeks of storage.

Tuberculosis of the Hip in Children.

H. R. MCCARROLL AND R. D. HEATH (*The Journal of Bone and Joint Surgery*, October, 1947) state that the ultimate aim for the optimum end-result in tuberculosis of the hip in children is arthrodesis of the hip. To accomplish arthrodesis in a young child early enough to prevent massive destruction of all bone structures in the region of the hip, and to prevent the extreme secondary changes in the extremity which result from prolonged fixation, is no simple task. The onset of the disease in children up to the age of five years is not uncommon; they usually have little resistance to the infection, and massive destruction in the joint follows very rapidly. Because of this fact and the early stage of bone development, the authors prior to 1940 maintained continuous and complete immobilization in plaster of Paris spicas until the child was aged eight or nine years. Of 43 hips immobilized from two to eight years in preparation for arthrodesis, satisfactory results following arthrodesis were obtained in 31 instances. Of 72 cases of attempted arthrodesis, follow-up studies over three to twelve years have shown satisfactory results in 75%. For the local lesion these results are not unsatisfactory; yet, for the whole individual, this form of treatment is poor, because of the extreme secondary changes which occur. Of 43 hips which were completely immobilized for from two to eight years 28 showed shortening of from two to seven inches. The degree of shortening, in some instances at least, seemed in direct proportion to the period of complete immobilization. The authors considered that extreme degree of shortening could not be due to destruction around the hip. X-ray examination of other epiphyses of the extremity showed premature closure of the epiphyses, with deformity of the epiphyseal plates and metaphyseal areas. Because these changes led to extreme degrees of shortening after prolonged immobilization, the authors decided to attempt earlier operative fixation in every instance in which the general condition of the patient would permit. Their present routine is to immobilize the young child in a plaster spica for six months, and to improve his condition as much as possible by adequate diet and by general supportive care. If the general condition is suitable at the end of six months, a Brittain type of arthrodesis is attempted or some other extraarticular type of fixation. The patient is again immobilized in a plaster spica for from six to twelve months, adequate general care being maintained, and is then allowed to become ambulatory with the aid of crutches, being permitted to use the hip and extremity increasingly. Even if the arthrodesis fails, active use of the extremity is allowed to prevent damage to epiphyseal structures by prolonged fixation. A second attempt at arthrodesis is postponed and use of the extremity is permitted and encouraged during the interval. If the patient's condition is not considered suitable for surgery, plaster fixation is maintained for six months to allow the acute phase of the process to subside, after which active use of the extremity and if possible weight-bearing is instituted. Deformities at the hip may follow, but these can be corrected more easily than those in the extremity as a whole, which result from prolonged fixation and inactivity.

Glutamic Acid and Mental Functioning.

HARRY BAKWIN (*The Journal of Pediatrics*, December, 1947) has reviewed the literature on the use of glutamic acid as a stimulus to mental activity. It was first tried in epileptics because, since it yields free acid, it was thought to be perhaps anticonvulsant, but instead its use resulted in greater alertness, happiness and mental stability. Rats fed on glutamic acid performed twice as well in maze tests as did controls. It was found to improve the performance of children and adolescents a small but significant amount in a variety of intelligence tests. It promises to be of value to children of retarded or borderline mentality where the rise of a few points in the intelligence quotient leads to an appreciable increase in the child's ability to benefit by school or special training and to cope with his school fellows. By encouraging parents it will improve parent-child relationships and lessen the emotional disturbances that so often exaggerate the plight of the slow child. To be of value it must be given continuously and in large doses, generally varying from six to twenty-four grammes a day. This dose bears no relationship to age, and sometimes causes gastric distress.

Addis Count in Acute Nephritis.

MARGARET D. GILES (*Archives of Disease in Childhood*, December, 1947) describes the Addis count of urinary sediment, and from a review of the literature and from her own observations defines the normal limits for casts, red cells and white cells in the twelve-hour specimen of urine. She considers that the red cell Addis count gives as much information as the full Addis count, and by saving a great deal of work makes the test of greater clinical use. In the twelve-hour specimen a total of 600,000 red cells is given as the upper limit of normal. The author examined 218 patients with acute nephritis by Addis counts and routine renal function tests. Of these 41 were found to pass into a latent stage of the disease, and of the 41 the raised Addis count was the only sign of persisting disease in 25 cases. In the majority of patients the Addis count returned to normal within an average of sixteen weeks. It is suggested that the Addis count gives a better indication of the prognosis in acute hæmorrhagic nephritis than any other test of renal function.

Congenital Deformities due to Riboflavin and Vitamin A Deficiency.

A DEMONSTRATION of the work of Josef Warkany on congenital deformities was presented at the Fifth International Congress of Pediatrics (*Quarterly Review of Pediatrics*, August, 1947). He has shown that congenital malformations of the skeleton can be induced in about one-third of the offspring of female rats raised and bred on diets deficient in riboflavin. These malformations are prevented when the rats are fed diets supplemented by liver or crystalline riboflavin. The abnormal rats have short mandibles, protruding tongues, short arms and legs and syndactylism. The skeletons show shortening of the mandible, shortening or absence of the tibia, fibula, radius and ulna, and fusion of ribs and sternal centres of ossification. Congenital deformities of soft tissues were found in the young of female rats raised and bred on diets deficient in vitamin *A*. About 75% of the young had a retrolenticular membrane, often in association with other ocular deformities. In the chest of the young some fœtal conditions persisted and diaphragmatic herniæ were seen frequently on the right side. The kidneys were hypoplastic and horse-shoe kidneys sometimes formed. Persistence of Wolffian and Müllerian ducts side by side, retardation of the development of the uro-genital sinus and ectopic ureters also occurred.

Aqueous Preparations of Vitamin A.

J. M. LEWIS *et alii* (*The Journal of Pediatrics*, November, 1947) have investigated the use of an aqueous solution of vitamin *A*. The administration of this aqueous preparation brought about much higher blood levels of vitamin *A* in infants, children and adults than did an oily preparation. The fæcal excretion of vitamin *A* was determined in seven infants receiving vitamin *A* dispersed in an oily and in an aqueous vehicle. The amount of vitamin *A* excreted in the stools was considerably higher when the oily preparation was given. Thus, 38% of the vitamin *A* intake was recovered in the stools when the oily preparation was administered, as compared with an excretion of 7% following the ingestion of the aqueous product. These results, therefore, indicate that the oily preparation was poorly absorbed and, conversely, the aqueous preparation well absorbed, and explain the wide divergence of blood levels following both preparations. Experiments in guinea-pigs and in rats revealed the same findings, namely, higher blood levels and lower fæcal excretion of vitamin *A* following the administration of the aqueous preparation. Furthermore, the vitamin *A* storage in the liver was considerably higher when the

aqueous preparation was given. In conditions affecting the absorption of fat, as, for example, cystic fibrosis of the pancreas and obstructive jaundice, the administration of vitamin *A* dispersed in an aqueous vehicle brought about excellent rises in the vitamin *A* concentration in the blood, in contrast to the flat absorption curve obtained with the use of an oily preparation. The oily product contained vitamin *A* esters dispersed in fish liver oil, whereas the aqueous preparation consisted of vitamin *A* alcohol dispersed in an aqueous solution of propylene glycol containing vitamin *B* components, ascorbic acid, and a "solubilizer", mannitan monolaurate. Experiments carried out in rats, in guinea-pigs and in children indicated that the superiority of the aqueous preparation was not due to the presence of vitamin *A* alcohol, vitamin *B* components, or ascorbic acid, but rather to the dispersion of vitamin *A* (by means of the "solubilizer") in an aqueous propylene glycol vehicle.

The Effect of X Rays on Epiphyseal Growth.

J. A. REIDY, J. R. LINGLEY, E. A. GALL AND J. S. BARR (*The Journal of Bone and Joint Surgery*, October, 1947) state that X-ray dosages of from 800*r* to 1200*r* were directed over selected joints of six weeks old dogs. The treated area included the joint epiphysis and adjacent metaphysis. The epiphyseal cartilage was profoundly affected, and retardation of growth occurred. The authors point out that the articular cartilage of the treated joints showed frank microscopic evidence of degenerative changes, and was not so thick as in the controls. These changes were not sufficient to interfere with joint function during the period of the experiment, and were not apparent on gross examination. No significant abnormality was seen in the adjacent cortical or cancellous bone, synovial membrane, joint capsule, skin, or periarticular soft tissues. Mild bowing of the treated forelimb occurred in eight of twelve animals. No such deformity occurred in the hind limbs. Although shortening of the treated bone occurred in each instance, there was evidence of stimulation of growth at the untreated epiphysis of the treated long bone. The cause of this stimulating effect is not apparent. The authors state that the clinical use of X rays on growing epiphyses to control longitudinal growth inequalities in children must be considered as experimental and fraught with some potential dangers. Whether the danger is greater than is incurred in surgical procedures, such as lengthening or shortening of the lower extremity and epiphyseodesis, must await further investigations.

ORTHOPÆDIC SURGERY.

Recurrent Posterior Dislocation of the Shoulder.

J. C. R. HINDENACH (*The Journal of Bone and Joint Surgery*, July, 1947) states that the diagnosis of primary posterior dislocation of the shoulder is often missed, as the disability may not be severe, and the range of motion may be only slightly limited. Palpation of the head of the humerus below the spine of the scapula and the presence of an abnormal hollow below the acromion are the salient features. Antero-posterior X-ray pictures are very misleading, because they may show a normal outline of the shoulder joint. Lateral views through the chest, or supero-inferior views through the axilla, display the abnormal position of the head of the humerus. A shoulder may be dislocated posteriorly by trauma, and, although adequate reduction may be performed, the dislocation may recur every few months following specific strain. This is a true recurrent type. However, following an original dislocation, the disability may recur every time the arm is abducted, or flexed and elevated. Such a condition, although recurrent, is also habitual. True habitual posterior dislocation of the shoulder, however, often occurs in young adults without history of primary injury, and it is commonly bilateral. The author believes that usually in the true recurrent type of posterior dislocation which is due to trauma there is a detached *labium glenoidale*. Bankart's method of repair gives good results. A surgical approach is described which provides an excellent view of the entire posterior aspect of the shoulder capsule. When trauma has been the cause of the lesion, the capsule with *labium glenoidale* will be found detached from the posterior margin of the glenoid fossa. A method similar to that used by Bankart for anterior recurrent dislocations may be used to repair the lesion. The posterior surface of the glenoid neck and rim is "rawed", and four or six drill holes are bored through the rim. The capsule is reattached to the scapular neck by mattress sutures through the drill holes. The flexed arm is bandaged to the side for six weeks, with the wrist and fingers left free. Active movements are then encouraged. In the voluntary or habitual type of recurrent dislocation no detachment of the *labium glenoidale* may be found. Often a general bulging of the capsule is observed. The capsule should be incised vertically one-third of an inch from the glenoid so that the joint can be inspected. If no further abnormality is seen, some posterior support to the joint must be provided by using a "reversed Clairmont operation" or a posterior bone graft. The author believes that the old unreduced dislocation (the constant, permanent type) forms a good bony buttress for itself on the dorsum of the scapula. Limitation of shoulder range is not great and treatment is unnecessary.

Bone Grafts.

LEONARD F. BUSH (*The Journal of Bone and Joint Surgery*, July, 1947) states that regeneration in bone-grafting takes place only when the grafts are in contact with living bone; this occurs chiefly by metaplasia of connective tissue cells and has been demonstrated in autogenous and homogenous bone grafts as well as in simple fractures. Grafts have proved to be more successful in the young than in the aged. Sulphonamides inhibit bone calcification. Cortical bone is best for stability, while cancellous bone is preferred for osteogenesis. Blood types, according to the author's studies, have no bearing on the success or failure of the bone graft. In a series of 67 operations in which homogenous bone was used the post-operative course was not influenced by the compatibility or non-compatibility of the blood groups. In identical operations the morbidity of patients who had autogenous or homogenous grafts, or both, was similar. The Rh factor has no bearing on the results in the use of homografts. The author states that bone may be obtained from other patients upon whom operations are being performed simultaneously, or from relatives who are admitted to the hospital for this purpose; it may be transferred directly from the donor to the recipient. Delayed grafting, whether autogenous or homogenous, is the storing of bone in the tissues of a patient for use in secondary operations. This method has been used successfully on several occasions, but should rarely be necessary in institutions where a method of keeping a supply of bone in a bank has been instituted. Refrigeration is a convenient method of preservation of bone. Bone may be stored under sterile conditions at a temperature of 2° to 5° C. for periods up to three weeks and at −25° C. for an indefinite period of time. With both types of refrigeration, the bone is warmed to room temperature before being used. The author describes the technique of storage.

Osteochondritis Dissecans of the Talus.

R. B. RAY AND E. J. COUGHLIN (*The Journal of Bone and Joint Surgery*, July, 1947) discuss the ætiology, diagnosis and treatment of *osteochondritis dissecans* of the talus. They consider the condition due to trauma—either a single recognized episode or repeated minor unnoticed injuries—with localized vascular diminution leading to avascular separation of the loose fragment of cartilage and subchondral bone. The usual presenting symptoms are pain and tenderness at the site of the lesion; swelling and intraarticular effusion may occur. Pain and swelling subside with rest and elevation of the leg; but tenderness usually persists indefinitely. Joint motion is usually within normal limits, and physical examination in early cases may reveal no abnormality. Although X-ray examination is the principal method of diagnosis, routine antero-posterior and lateral views often fail to demonstrate the lesion, probably because the supero-lateral or medial pole of the talus, which is the usual site of the lesion, may be hidden by the superimposed lateral or medial malleolus. However, careful oblique projections will demonstrate the lesion. In eleven of the authors' fourteen cases the lesion was on the medial border of the superior articular surface of the talus, while in the remaining three it was on the supero-lateral articular surface. Seven of the patients had had either severe sprains or recurrent sprains of the involved ankle; two patients had had direct trauma. The duration of symptoms ranged from two months to four years, the average being 21·4 months. The authors consider that a patient presenting a typical history of repeated sprains, with localized tenderness over the anterior aspect of the talus and normal X-ray findings, should be followed carefully, since subsequent X-ray examination may disclose the lesion. Treatment should consist of excision of the loose fragment and saucerization of the remaining defect in the articular surface of the talus. The surgical approach to the ankle joint should be considered carefully, and should be chosen only after X-ray study and determination of the exact location of the lesion.

Special Article.

WESTERN AUSTRALIA AND ITS CAPITAL.

Those who visit Western Australia for the medical congress of 1948 will not perhaps be impressed at first by the enormous size of the State. This is an aspect of the country that does not hit you in the eye. You have to look at a map and read some statistics to discover how big a country you are visiting. There you will note that Western Australia is about a third of Australia. It is three times the size of New South Wales and about eleven times the size of Victoria. Superimpose a map of Great Britain (drawn to the same scale) on a map of Western Australia and you will marvel at the relative littleness of the island home from which men and women came to take possession of this vast territory.

All this is patent to the eye of the beholder—of maps; but not to the eye of the casual visitor to a medical congress. You would have to stay for months, travelling all the time, to get a vivid idea of the huge area of our territory. You might begin by taking ship at Eucla and steaming round to Wyndham—a distance of some 4000 miles—stopping at various ports for trips into the interior; such an expedition might give you a notion of the immense tracts of land still awaiting real occupation in the State of Western Australia.

The visitor from the eastern States does, of course, realize, whether he travels by train or by plane, that from the border to Perth is a considerable distance. He does get the impression of remoteness from the rest of Australia. But this impression is not nearly so deep as it was when I first came to Perth, thirty-five years ago, when a sea voyage of a week or so was the only means of getting here from Melbourne. Then indeed we did feel that we were coming to a distant island. Australia was cut in two as completely as if the intervening desert was an ocean.

What does seem likely to impress the visitor who comes to Perth, across the Nullarbor Plain, is the flatness of the country. It is true that before he reaches Perth he will have to cross the Darling Range; but this is only the broken edge of a plateau, not a range of mountains. There is a vast variety of scenery in Western Australia, but none of it Alpine. A small acclivity from which you can get a good view of the city of Perth has been named Mount Eliza, which shows that our early settlers had a remarkable capacity for making a mountain out of a molehill. Mount Bruce, away up in the north-west, rises to a height of a little over 4000 feet; Bluff Knoll, in the Stirling Ranges, near the south-west corner of the State, does not reach the 4000 mark. Nowhere in the near neighbourhood of Perth is there anything that can be called a mountain. Professor W. A. Osborne, of Melbourne, once wrote an interesting essay on "The Physiology of High Altitudes". Rereading it, one notices that he does not think any altitude of less than 4000 feet worth mentioning for its effects on the human frame. Perth is one of the most beautiful cities in Australia; but its citizens sometimes think, with vain regret, how much more beautiful it would be if there was a Mount Wellington in the background of the picture. And this is not merely an æsthetic regret; our climate is such that a week or two in the bracing air of a mountain resort would be invaluable for everyone who cannot afford a trip to distant lands.

On the whole, however, dwellers in the south-west—the only part of the State which visitors to the medical congress are likely to see—have every reason to be thankful for their climate. It is very pleasant and very equable. He would be hard to please who should ask for a better climate than that

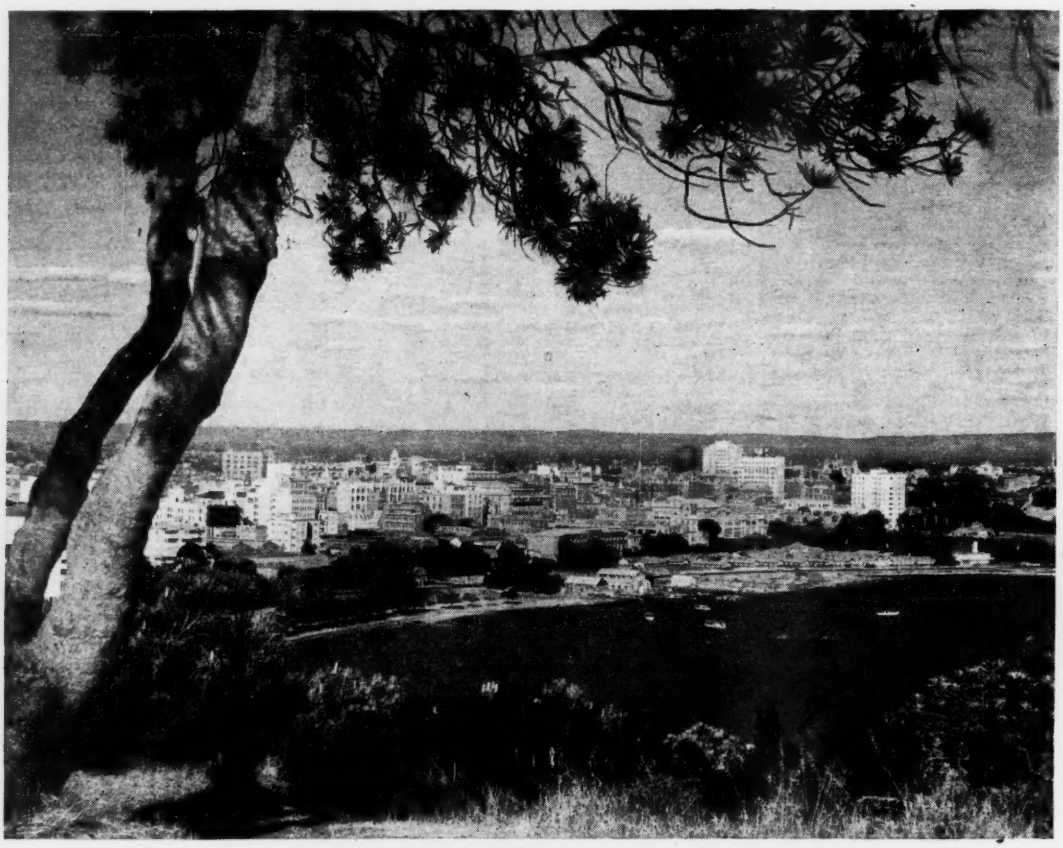


Figure I: View of Perth from King's Park.
By courtesy *The West Australian*, Perth.

of Albany, where on the hottest night of summer (with rare exceptions) one sleeps under a blanket and not under a heap of blankets on the coldest night of winter. Perth, over 300 miles north of Albany, is not quite so happy in the three hot months of the year. But the heat, unpleasant while it lasts, seldom lasts for more than a few days without a break; and the breaks have not the explosive suddenness of a Melbourne break. On the whole, a delectable climate. It is, however, a climate in which a tendency to take things easily is apt to make itself felt. The climate of Kalgoorlie, nearly 400 miles inland, is much less agreeable than that of Perth—far hotter in summer and far colder in winter—but, summer and winter alike, more bracing, less conducive to languor. This characteristic of the Perth climate has been the source of many gibes, probably invented by Perth citizens themselves, such as the time-worn saying that the initials "W.A." stood for "Wait Awhile". It used to be said that if you dropped a sovereign (in the days when there were sovereigns to drop) in a Perth thoroughfare, if you remembered the spot you could return next day and retrieve the coin; such was the incorruptible honesty of the citizens of Perth. But cynics said that if this was true it was not because the citizens were too honest to pick up the coin, but because they were too lazy. There was probably a grain of truth in these jests. When, after the banking collapse in the east, an army of adventurous spirits came pouring into this State at the call of gold, they found here a city which they considered somnolent. If the "Roaring Nineties" have been rightly named, the roar was a new noise in the ears of the citizenry of Perth. The earliest pioneers of the State were, many of them, brave, adventurous, enterprising, of a tremendous energy; no one can deny it. But neither can anyone deny that, when the nineties brought that wave of gold-seekers foaming into this peaceful land, they found a small, happy and easy-going community, more or less basking in the pleasant sunshine. They brought into it an element of feverish hustle which the inhabitants disliked and resented. For many years there was a coolness—we shall not call it a feud—between the original "gropers" and the "t'other-siders". The quarrel has long since died down. Perhaps the softness of our climate has helped to mitigate ancient acerbities.

Perth is, as I have said, one of the most beautiful cities in Australia. Its beauty, like the beauty of Sydney, comes mainly from the stretches of water it enfolds. The visitor should be conducted, as soon as possible after his arrival, to King's Park, on the bit of rising ground already alluded to. It is not a mountain, but it is high enough to give you a view of the central part of the city, lying spread out before you. If the hour is sunset, I maintain that you will have a vision unsurpassed if not unrivalled by anything in any other of our capital cities. It sometimes looks like a city of fairyland.

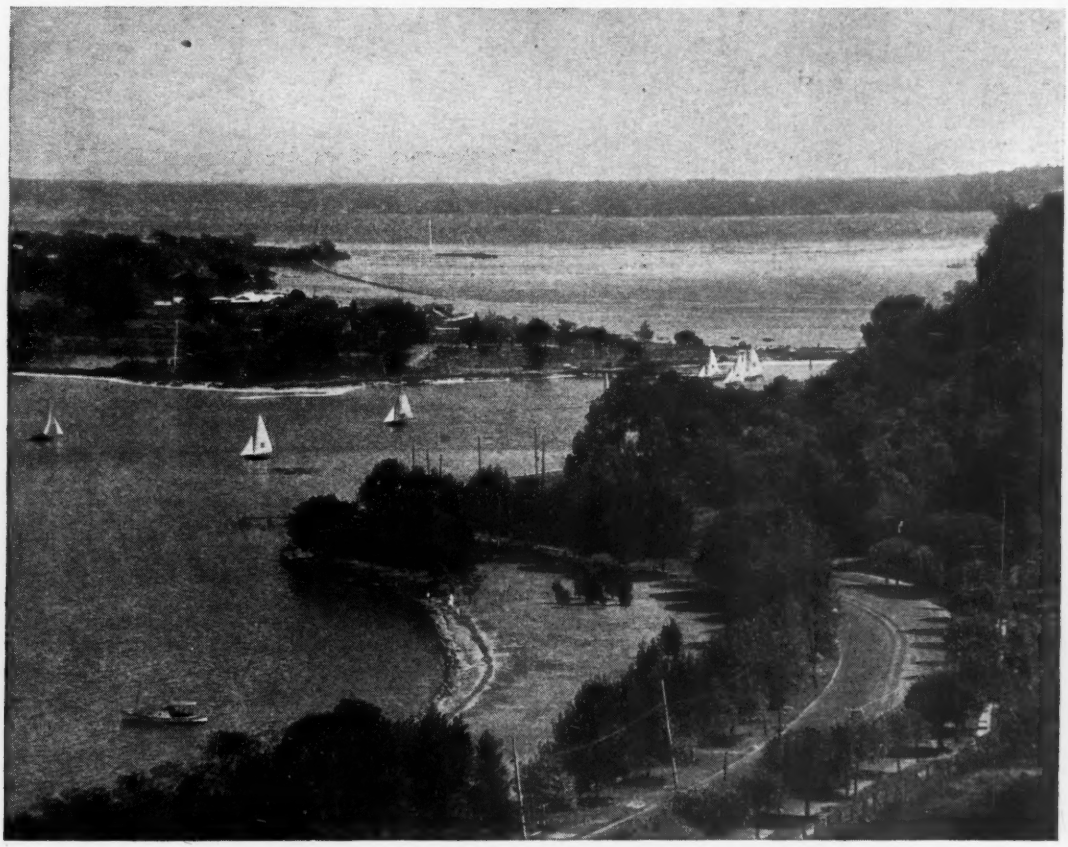


FIGURE II: Showing the confluence of the Canning and Swan Rivers.
By courtesy *The West Australian*, Perth.

When you go down into the city itself you will find, I think, no very marked difference from other Australian cities and very little that is distinctively Australian. (All Australian cities are more or less plagiarisms from the cities of the old world, and especially the cities of Britain.) One or two of the streets are broad; most of them are regrettably narrow. Some of the architecture is good; for some of it the only remedy is a bomb. The building which will perhaps stay longest in the visitor's memory is the old Town Hall, which was designed by a famous English architect. For the rest, the public and private buildings have nothing distinctively Western Australian about them. The differences between our capital cities are to be found, if at all, in the spiritual atmosphere, the moral colour, the prevailing social note of each; not in anything visible to the naked eye of a visitor in a hurry. And what applies to the cities applies to the States as wholes. Each has its own spiritual climate.

This may some day be the theme of a subtle sociological study.

Meanwhile the observant visitor may feel that a friendly spirit pervades the life of Perth, which is lived, as I have hinted, at a somewhat more leisurely *tempo* than in the great cities of the eastern States. Wealth—though it is hard to find statistics on the point—appears to be better distributed here than elsewhere. There are no millionaires and no slum-dwellers. Perth has no ruffianly underworld—which is not to say that it has no individual ruffians. There are criminals, but not "pushes". The moral tone is, we can say without pharisaical complacency, better than in some other cities of Australia. The racketeer is less in evidence here. There is a higher general standard of decent behaviour.

Figure III: St. George's Terrace, Perth.
By courtesy *The West Australian*, Perth.

Here, as elsewhere, however, there is the problem of the drift to the city. The population of Perth—counting Fremantle as a part of Perth—was at the latest census 265,000, which means that considerably more than half the population of the State is concentrated in these few square miles. This is more or less true of all Australian capitals except Hobart. How to make life in the country more attractive to young men and women, compared with life in the city, is Australia's riddle of the sphinx. We are thought of, by the rest of the world, as a nation of farmers and squatters; in reality we are the most urban nation in the world. Nowhere in Australia is this fact so conspicuous as in Western Australia. Its immense size emphasizes the problem. The isosceles triangle which has Kalgoorlie at its apex and Geraldton and Albany at the tips, and Perth in the middle of its base—this triangle, with a number of country towns in it, may be called the inhabited part of the State; and if you look at the map you will see what a small corner it is. The enormous territory to the north and the east consists mainly of the famous "vast empty spaces". Visitors who have crossed the Nullarbor Plain will have had a glimpse of one of these spaces and will have wondered whether it will ever be other than vast and empty. But there are yet vaster spaces to the northward in which we know the soil to be fertile and the climate good. We in Western Australia are growingly conscious of this immense unoccupied background. It has been brought home to many Western Australians by visits to a near neighbour. Before the war it was common for them to spend a holiday in Java—to pass from the most sparsely populated country to one of the most densely populated countries in the world. Perpend the following figures. Java has a population of about 44,000,000 and an area of 48,000 square miles. Victoria, nearly double the size of Java, carries a population of a little over two millions. Western Australia, with an area of nearly 1,000,000 square miles, has a population of nearly 500,000. In other words, Java carries about 1000 persons per square mile; Western Australia carries almost exactly one person per two square miles. It is no wonder that in Western Australia we are more acutely aware than elsewhere in the Commonwealth of the emptiness of the land and of the need of immigrants.

Another perceptible difference between the mental atmosphere of this State and that of the rest of Australia is our keener awareness of our own history. We are nearer to it—nearer to the adventurous pioneering days. In Victoria it is not possible to learn at first hand what happened at the Eureka Stockade; but here visitors to the congress may quite probably meet elderly persons who joined in the wild rush to Coolgardie and who will tell many picturesque, though possibly inaccurate, stories of exciting incidents on the goldfields. The days of the gold rush are still a vivid memory. The days of the pioneers can hardly be called a memory, for they are not yet over. Middle-aged pioneers are to be seen walking the streets of Perth, when they come down from the cattle country of the far north for a holiday in the city, indomitable men who drove their stock into

unknown, unexplored country, and built homes for themselves and their families in far-off places. I fancy it is not so easy to meet pioneers of this type in other States. History comes closer to us—the history of the making of our country.

This may be illustrated by the mention of a single personality, a man of remarkable and dominating qualities. In King's Park, in a commanding position, you will see Bertram Mackennal's statue of Lord Forrest. First becoming known as an intrepid explorer, John Forrest made himself a political leader and was for many years, before federation came, practically a dictator. He was not, as I read him, a man of high intelligence; but he had tremendous will-power and robust common sense. When he had made up his mind that a thing had to be done, nothing would turn him aside; he made straight for his goal. He had a masterful way with him. No man ever dominated any other Australian State as Forrest dominated Western Australia before it became a part of the Commonwealth. Afterwards, when he entered Federal politics, he was overshadowed by men of more supple intelligence and greater breadth of mind. But in his own State he is still remembered by some of his achievements, such as the Kalgoorlie water scheme; and Perth has no suburb without its Forrest Street. But many now living remember him personally, with his burly figure and his bluff manner, and his simple, downright language. That illustrates what I have just said about our history being close to our consciousness.

Though Forrest cut such an imposing figure in his day, no one has yet thought it worth while to write a biography of him. There is another biography which remains to be written: that of John Winthrop Hackett. Hackett was not in any sense a captain of men as Forrest was; but he was a man of far more penetrating intellect; he was indeed the brain behind much that Forrest did or essayed to do, his counsellor and inspirer. Having become, after many vicissitudes, the owner and editor of Perth's leading newspaper, he was for many years the chief shaper of public opinion throughout the State. Forrest did many things which he could not have done without Hackett's backing.

There was one matter about which Hackett felt strongly and Forrest did not feel at all: higher education. A university man himself, Hackett was bent on establishing a university in Western Australia; Forrest appeared to regard this as one of his friend's harmless fads, for which the time was not yet ripe. Hackett had his way, and his chief memorial, as he would have wished, is the University of Western Australia, of which he was the first chancellor and which he nursed through its precarious infancy. Unhappily he died too soon to see the beautiful buildings which the wealth he bequeathed to it provided for it.

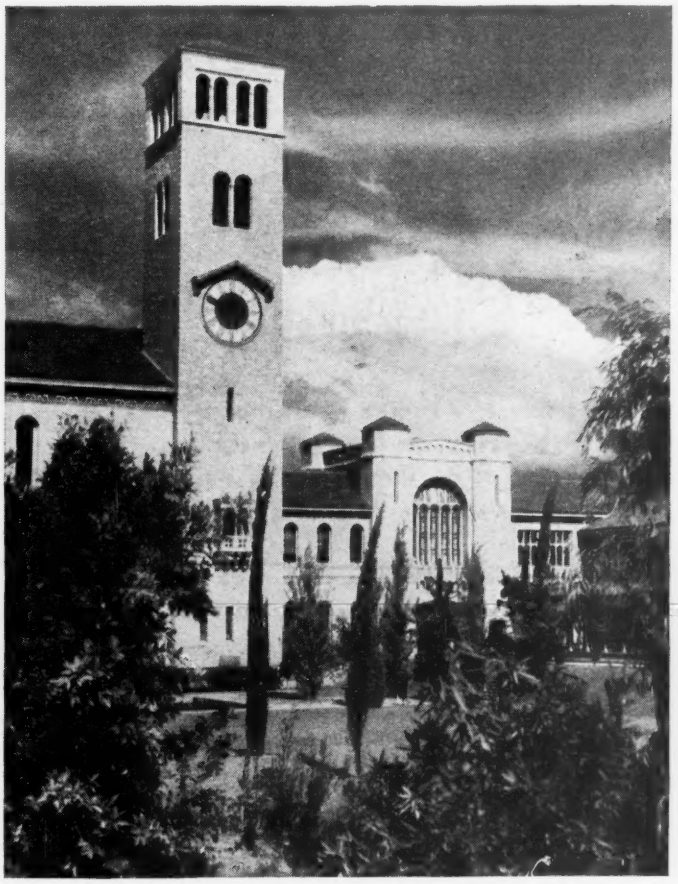


FIGURE IV: The University of Western Australia.
By courtesy *The West Australian*, Perth.

As the medical congress will see much of these buildings, it would be superfluous to describe them here. They were the crowning work of a young architect of genius, Rodney Alsop, of Melbourne; it comforts his friends to know that he, at least, did live just long enough to see his dream take shape in stone. What he designed is, our visitors will admit, the most beautiful, outwardly, of all Australian universities. It is not too much to say that by this achievement Alsop raised the standard of architectural taste throughout the community, and that the nobility of the buildings has helped to win a respect for the university and all that it stands for. My private opinion is that the University of Western Australia plays a greater part in the communal life than any other university in Australia, and that many citizens would feel no shock of surprise if they found Perth described, in a textbook of geography, as a university city.

It remains only to add that the university is at present in the throes of an effort to establish a medical school. Hitherto students of medicine have had to get their training in the Eastern States, where the universities are now overcrowded. The argument for a medical school in our own university has become overwhelmingly strong. The only obstacle is finance. It would be better for students to be sent abroad for their training than to be given a tenth-rate training at home. To secure a first-rate teaching staff and first-rate equipment costs more than we have so far believed ourselves able to afford. But the public is beginning to see that there could be few better investments for public money. We all hope the approaching medical congress will help to strengthen the hands of those who are fighting for the establishment of our own medical school.

WALTER MURDOCH.

The attention of readers is directed to the leading article, to "Congress Notes" which appear on another page, and to the coloured illustration of Western Australian wild flowers. For the coloured illustration we are indebted to the Government Tourist Bureau of Western Australia and for the photographs of Perth to *The West Australian*, Perth.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at Wangaratta on October 25, 1947, the President, DR. A. E. COATES, in the chair. The meeting took the form of a series of clinical demonstrations by members of the staff of the Wangaratta District and Base Hospital, followed by a paper read by DR. A. M. HILL.

Splenic Anæmia.

DR. F. R. PHILLIPS showed a healthy looking girl, aged thirteen years, who had been subjected to splenectomy for splenic anæmia. He said that she was the only member of the family who had had splenomegaly or serious anæmia. Her tonsils and adenoids had been removed some years earlier, but at ten and a half years of age the girl had vomited after epistaxis and the vomitus was slightly blood-stained. She was in the care of the late Dr. Kelly, who had ascribed the blood-staining to the preceding epistaxis. A year later, however, the girl had vomited a lot of blood, and three days afterwards a direct transfusion of blood was made from a compatible donor. Dr. J. P. Major had been in Wangaratta at the time, and, in consultation with Dr. Kelly, had diagnosed the condition as splenic anæmia; the spleen was moderately enlarged and the diagnosis was supported by the report on a complete examination of the blood. Shortly afterwards, in October, 1945, Dr. Kelly had removed the spleen, and Dr. Cole, who was present at the operation, had since informed Dr. Phillips that the splenectomy was simple and straightforward. Since then the girl had grown a little and had remained well until in October, 1946, she had had abdominal pain for four days. Dr. Phillips said that he had attended her and had ascertained that the hæmoglobin value was 80% and that the blood count was reasonable, though, as a precaution, he had instituted iron therapy. She made a good recovery and resumed attendance at school, but two weeks before the meeting another severe hæmatemesis had occurred. He had arranged her admission to the hospital and found that the hæmoglobin level had fallen to 48%. After a transfusion of citrated whole blood the hæmoglobin value rose to 81%, and, as no further bleeding followed, she was discharged from hospital. Dr. Phillips asked for opinions as to further management and, in particular, wished to know whether a porto-caval anastomosis should be attempted.

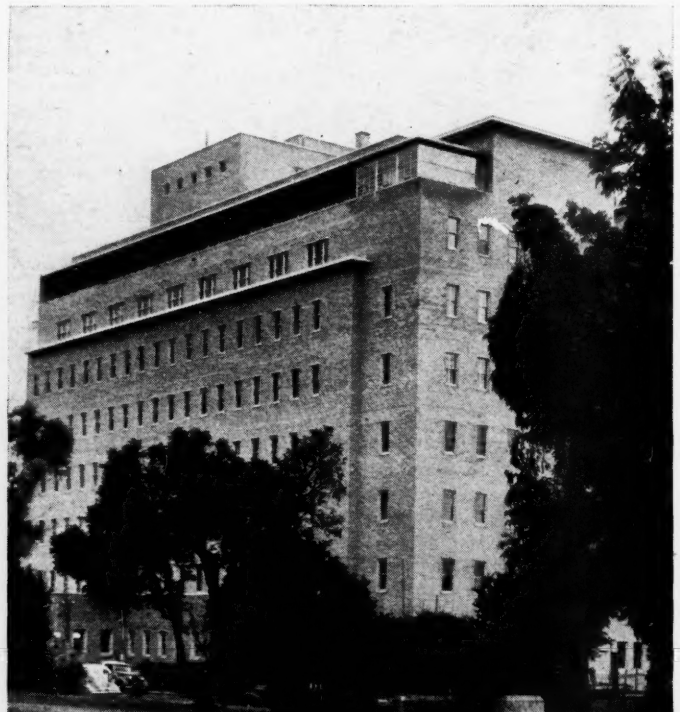


FIGURE V: The Royal Perth Hospital. (First section of the new hospital.) By courtesy *The West Australian*, Perth.

DR. H. BOYD GRAHAM said that, as the latest report on the blood examination was satisfactory and as the girl was strong, healthy and active, he thought that it was not necessary to do anything beyond keeping her under observation and following the state of the blood by periodical investigations. The prognosis in splenic anæmia, however, was not good, as the portal cirrhosis was progressive and the risk of fatal hæmatemesis was ever present; the parents should be warned of that risk to enable them to obtain prompt attention if bleeding started. Transfusion of blood was of paramount importance, with as little delay as possible. In the normal course of events sooner or later one could predict that the disease process would enter the stage known by the name of Banti, the main features of which were advanced portal cirrhosis, ascites, grave anæmia and leucopenia. Dr. Boyd Graham said that he would prefer some surgeon present to discuss what might be done operatively for her; it could conceivably be advisable to carry out some procedure such as omentopexy or porto-caval anastomosis while the child's condition was good, rather than to embark on surgical interference as a last resort.

DR. A. E. COATES said that the operation of porto-caval anastomosis was not yet established as suitable for splenic anæmia in childhood, and as the girl was well no harm would be done by waiting for reports of further experience of the operation and its aftermath.

Cœliac Disease.

DR. VERNON DAVIES showed a plump demonstrative little girl, aged four years, who had been under treatment for two years for cœliac disease. He said that the child had first come under notice in December, 1944, when she had sustained an electric burn that took three weeks to heal. At that time it was noted that she had frequent loose stools, but that aspect of the case was not investigated till she was brought to Dr. Davies in an emaciated state in November, 1945, suffering from acute bronchitis, pneumonia and *otitis media*, and passing large fatty stools. As there was a history of contact with tuberculosis and as there was positive radiological evidence of lung disease a tuberculous ætiology was at first suspected. Dr. Davies had sent her to the Children's Hospital on January 19, 1946, as an example of cœliac disease and that diagnosis was confirmed there. She was very ill indeed and near the point of death, but recovered sufficiently to be discharged from the hospital and returned to her home in April, 1946. In May and June, 1947, she was again very ill with great enlargement of the abdomen and inability to walk for eight weeks. There was a fast response to treatment by injections of "Anahæmin" and oral administration of "Vibex"; the weight had not exceeded twenty-three pounds from November, 1945, to August, 1946, but she gained an additional seven pounds in four weeks, and since then had been materially improved in nutritional status. In May and June the fat content of the fæces was approximately 50% of total fæces and the split fat amounted to 25%. Recent estimations showed improvement in August, but further deterioration since then. The latest report stated that the fat content was 62% and the split fat 32%. Dr. Davies asked for opinions as to whether intensive treatment should be continued. He added that since May, 1947, the child had had four convulsions and was taking phenobarbital regularly. He would also like to know whether dietary restrictions should be enforced.

DR. H. BOYD GRAHAM said that the child seemed to be mentally bright, but was over-fat, and her behaviour indicated that she was pampered and over-indulged. He thought that there were clear indications for careful dietary with considerable reduction of fatty and starchy

items and compensating increases in protein food with unlimited fruits and vegetables. She should not be underfed. The diet should adequately cover her caloric requirements, but its composition should be carefully considered. He did not think that intensive treatment by injections of "Anahæmin" should be repeated at present, but vitamin concentrates such as "Vibex" and "Halivol" should be continued. In conclusion, he contrasted the favourable prognosis for the child in spite of unprepossessing present circumstances with the unfavourable prognosis for the patient previously shown with splenic anæmia, although she looked the picture of health and happiness.

Leucoplakia and Epitheliomata of the Mouth.

DR. J. B. MCMIKEN showed a male patient, aged forty-five years, who had come under his notice on October 16, 1946, with leucoplakia of the mouth of several years' duration. The patient was edentulous and the blood serum had failed to yield the Wassermann reaction. At the Alfred Hospital in 1940 he had had a radon implant for epithelioma inside the cheek. In August, 1946, the condition recurred inside the mouth in the cheek area, and by October it had spread onto the lower lip where it took the form of a hypertrophic epithelioma measuring 3·0 centimetres by 1·5 centimetres. There was also a large grey area of leucoplakia, 4·0 centimetres by 3·0 centimetres in size, on the left side of the hard and soft palate. Initial treatment given by Dr. McMiken had consisted of a fairly large radon implant (13 needles 6·5r). By January, 1947, the treated area was well healed and a biopsy specimen taken from the leucoplakic area on the soft palate proved to be non-malignant.

In July, 1945, a recent area of leucoplakic change with areas of hypertrophic growth and epitheliomatous degeneration was evident along the inside of the right cheek at the level of the artificial denture. At that stage the patient was seen by Dr. Hands, and Dr. McMiken effected another radon implant, putting in eight needles right round the soft palate. The area in the right cheek had healed completely, but the leucoplakia on the left side of the soft palate was still present.

In summarizing the case for discussion, Dr. McMiken said that it was an example of severe leucoplakia with two separate foci of malignancy treated by radon with satisfactory results. He considered it unwise to treat leucoplakia *per se* by radiation; the treatment would clear the leucoplakia temporarily, but the lesion recurred later. It was wiser to leave it alone until malignant change intervened and then that could be irradiated by the use of moderate dosage. Epitheliomata occurring in leucoplakic areas were slow to metastasize to glands, so the glands need not be treated until they in turn were malignant and then irradiation offered the best prospects in his opinion.

At the request of Dr. Coates, Dr. McMiken showed another patient and a discussion took place on the two cases.

Melanoma.

Dr. McMiken then showed a female patient, aged eighty years, who had come under his care on January 3, 1945, having had a pigmented spot on the back of the lower portion of the left calf for very many years. In April, 1944, a lump which ulcerated appeared in the centre of the pigmented spot. In October, 1944, a pigmented spot appeared on the back of the left forearm and a nodule came at its centre. The larger spot measured 4·0 by 3·0 centimetres; the nodule had a diameter of 1·7 centimetres and was raised 1·0 centimetre. The larger one was bleeding freely. A rodent ulcer was also present on the right side of the neck. Dr. McMiken said that, though it was recognized that melanomata were not very radiosensitive, he had used a circle of nineteen needles with eight seeds in the raised area on the leg and six needles in the left forearm; that was close to the limit of tissue tolerance. The reaction was rather severe, but the forearm healed in two months. The leg healed partially and then radionecrosis occurred. Both areas had healed a year afterwards, but since then the leg area had broken down again and a painful necrotic ulcer had formed. Radiotherapists considered that it was better to irradiate than to excise a melanoma in such a case, and, considering that a metastasis was present when treatment was undertaken, he thought that it was satisfactory to have the old lady alive and fairly well except for the painful ulcer three years afterwards.

DR. A. E. COATES said that he had encountered instances of severe pain associated with implantation of radium needles in the mouth and also necrotic ulceration that would not heal; the periangiitis prevented the normal process of repair. Whilst radium was a wonderful reliever of the lesion, it was liable to cause pain later on. As the patient was elderly and had become used to her ulcer, he advised that no further treatment was indicated beyond the use of *Unguentum Renaglandin Anæsthetica* to mitigate the pain. Operative excision was the only cure for the necrotic ulceration, and he would advocate it in a younger patient. The old lady, however, would need to be in plaster for months after an operation and would run the grave risk of hypostatic pneumonia.

Rheumatoid Arthritis and Osteoarthritis.

DR. A. J. M. WHITE showed a married woman, aged forty-seven years, who had undergone treatment for rheumatoid arthritis. She was the mother of three children, the youngest of whom was aged eighteen years. She had had signs of rheumatoid arthritis for twenty-three years, the illness commencing shortly after the birth of the first child. The joints originally involved were the metacarpo-phalangeal ones and then the ankle joints and the right temporomandibular joint. Later in succession the knees, wrists, elbows and shoulders were affected. All the joints were swollen and tender and there was transient loss of power. In the earlier years she had remissions while pregnant and exacerbations shortly after confinement, and on many occasions since she had had to stay in bed because of the painful crippling. She came under medical care in Wangaratta in 1941. Previously the treatment had been palliative, consisting of baths, liniments and mixtures. In June, 1942, chrysotherapy was first used by Dr. Vernon Davies, and courses of treatment had been given by Dr. Davies or by Dr. White subsequently in June, 1944, December, 1946, and June, 1947, after each of which she felt very much better.

Dr. White drew attention to the informative article by L. J A. Parr and Eva A. Shipton, of Sydney, published in THE MEDICAL JOURNAL OF AUSTRALIA, March 15, 1947, page 323, and said that for six weeks before the meeting the patient had had three tablets daily of sulphadiazine. In the earlier weeks she was very languid, but had improved greatly in the past week. She had had a couple of "turns" during the course of treatment with excessive tiredness followed by palpitation of the heart, dyspnœa, faintness and absolute loss of power and inability to speak. The duration of each attack was approximately ten minutes. While taking the sulphadiazine she also received "Polyhæmin" tablets and vitamin therapy. Dr. White detailed the normal findings on examination of the blood by hæmoglobin estimations, erythrocyte and leucocyte counts, study of the films and differential counting. That procedure was carried out on October 1, 1947, and again on October 16, 1947, and October 24, 1947. The patient's blood was classified in group B (3). The blood sedimentation rate on October 6 was ten millimetres in one hour and on October 24 was fifteen millimetres in one hour. On October 14 Dr. McMiken demonstrated normal electrocardiographic tracings in all four leads.

At the time of the meeting there was limitation of movement at the elbows, wrists and small joints, but those present were assured that definite improvement in range of movement had taken place. The right knee was swollen, but the other joints were practically normal. The pulse rate was only 80 per minute and the blood pressure readings were 110 millimetres of mercury (systolic) and 70 millimetres (diastolic). In other respects the clinical examination was negative. Dr. White said that he had presented the patient and her history to show that gold injections had a definite place in the treatment of rheumatoid arthritis, and to draw attention to new forms of treatment such as the use of the sulphonamides and the transfusion of blood from pregnant women as advocated by Barsi, of Budapest, in the recent article published in the *British Medical Journal* of August 16, 1947.

Dr. Coates invited Dr. McMiken to show his patient suffering from osteoarthritis and suggested that afterwards the two arthritic conditions would be discussed.

DR. J. B. MCMIKEN showed a male patient, aged sixty-four years, with bilateral crippling osteoarthritis of the hips who had been treated with injections of dilute lactic acid. He had come under observation in July, 1947, having had osteoarthritis of the right hip for many years with almost complete limitation of movement at the hip joint. Similar trouble had recently affected the left hip joint and the constant aching had rendered him miserable and sleepless and he was unable to walk further than one hundred yards. He was faced with the prospect of having to vacate his little poultry farm and become bedridden. His daughter, who was a trained nurse, had insistently asked that he should receive injections as described in articles in the *British Medical Journal*, such as "Mono-articular Osteoarthritis of the Hip and Treatment by Acid Injections", by

W. Grant Waugh, June 23, 1945. Achlorhydria was found to be present and it was decided to use injections of lactic acid 0·2% solution (pH 5·8-5·2). Three injections were made into each hip and into each knee joint. Technical difficulties were encountered in introducing the solution into the hip joints. It was desirable to fill the joint, and then on account of the pain to withdraw some of the fluid to ease the tension. The patient had improved considerably, though he was still very crippled. The principle applied was to reacidify the joint fluids which became alkaline in osteoarthritis. Dr. McMiken said that he presented the patient to attract attention to the lactic acid treatment and the gratifying result obtained.

DR. CHAS. STANLEY said that the woman with rheumatoid arthritis seemed to have been tided over the bad time, that the condition had burned itself out and that he expected that she would have less pain and more freedom of movement as time went on. Dr. Davies and Dr. White were to be congratulated on their management of the chrysotherapy, but the treatment was by no means devoid of danger; he had seen three patients who had developed chronic nephritis ascribable to the therapy. With reference to the lactic acid treatment of osteoarthritis, Dr. Stanley said that they had used it on four patients at the Bendigo Hospital with promising results; the field had opened for giving help where help was badly needed.

DR. L. J. GURRY said that he did not agree with Dr. Stanley's opinion that the rheumatoid condition was burnt out. The woman was a worrier, and, as was often the case with invalids, she was very wrapped up in herself and her ailments. He considered that the wrists and the fingers would be improved by means of hot paraffin wax baths. She had a dry scalp and fine seborrhœic condition and might easily get exfoliative dermatitis if any more injections of gold were given. On the man with osteoarthritis, Dr. Gurry's comment was that the result was remarkable.

DR. F. R. PHILLIPS expressed alarm at the incidence of chronic nephritis associated with injections of gold salts and mentioned that in association with Dr. Rohan he was treating a patient with arthritis with 0·1% solution of sodium acid phosphate. Most of the patients seemed to be fat ladies with deformed joints, and it was difficult to get the solution into the hip joint by using the localizing points taught as normal in surgical anatomy. He would like Dr. Rohan to describe the method that they had found most practicable.

DR. M. ROHAN said that he started by putting his finger on the femoral artery and introducing the needle half an inch outside the artery and half an inch below the fold of the groin. For the anterior approach he used a lumbar puncture needle and inserted it approximately three and a half inches, but a six-inch needle was necessary for the posterior approach. The capsule was very thick and it held the needle tightly. He first injected ten millilitres of "Novocain" solution and could feel the needle hit the neck of the femur. When the joint was entered he withdrew the plunger of the syringe and allowed the solution to drip back through the needle. He had found that he could not recover more than two millilitres out of the ten millilitres introduced. He then flushed the joint out two or three times with the sodium acid phosphate solution, replacing a few millilitres each time, and finally took off a little till the pain of tension disappeared.

(To be continued.)

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the Institute of Medical and Veterinary Science, Adelaide, on May 2, 1947.

The Brain in Amphibians.

DR. W. R. ADEY read a paper on examination of the brain in amphibians. He said that as part of a general survey of the excitable parts of the brain in the major vertebrate groups, stimulation and extirpation experiments had been carried out in a series of amphibian brains. The material used was mainly the giant canefield toad, *Bufo marinus*. The frogs *Hyla aurea* and *Lymnodynastes dorsalis* were used as a check. Unipolar thyratron stimulation gave the best results. The stimulus was of the order of one-quarter to one volt at an average frequency of forty per second.

Excitation of the forebrain and basal ganglia gave no motor response. The excitable part of the brain was found to be limited to the optic lobes of the mid-brain. There, movements could be evoked from all the limbs, from the eyes, and from the trunk and neck muscles. Movements of inspiration and expiration also occurred. The degree of somatotopic localization varied. Movements of the globe came mainly from the anterior pole of the mid-brain, and movements of the hindlimbs mainly from the posterior pole. Forelimb movements were evoked from most of the optic lobe.

It was found that control of the limbs was bilateral—each optic lobe controlled the limbs on both sides of the body. Division of the mid-brain and *medulla oblongata* in the midline eliminated contralateral movements. The movements produced by stimulation followed the general pattern of the movements used by the animal in swimming and hopping.

Stimulation of the brains of the frogs *Lymnodynastes dorsalis* and *Hyla aurea* gave similar results to those seen in *Bufo marinus*. The control of the limbs was bilateral, but movements of the eyeball rarely occurred.

A series of experiments had then been carried out to determine as far as possible the nature of the higher control of muscle tone in these animals. Transverse sections were therefore made at varying levels in the brain. Removal of the cerebral hemispheres caused no alteration in muscle power, nor in muscle tone. Transverse section through the middle of the mid-brain was followed by moderate forelimb rigidity. Section between the mid-brain and the cerebellum produced complete flaccidity.

Section immediately behind the cerebellum produced intense rigidity in the forelimbs and neck extensor muscles. Sections had then been made in a series of animals at gradually lower levels in the medulla. The rigidity diminished as the level of the section moved caudally, and finally disappeared in an animal transected at the spino-medullary junction. This distribution followed closely the outline of the vestibular nucleus of the eighth nerve.

Dr. Adey said that work was at present being conducted to determine the role of the cerebellum in the production of hypertonicity. These experiments had so far proved inconclusive.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Overseas Lecturer.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Dr. Earl R. Carlson, American authority on cerebral palsy, will give a post-graduate lecture on Wednesday, April 14, 1948, at 8.15 p.m., in the Stawell Hall, 145, Macquarie Street, Sydney, to which medical practitioners are invited.

On Saturday, April 17, a clinical meeting with case demonstrations will be conducted by Dr. Carlson at the Spastic Centre, Queen Street, Mosman, at 10 a.m., to which members of the Sections of Neurology, Psychiatry and Neurosurgery, Orthopædics and Pædiatrics are invited. As the accommodation is limited, Section members who intend to be present are requested to notify Dr. C. Burton-Bradley's secretary at the Spastic Centre no later than April 12, 1948. Telephone: Number XM 3027. Other members of the medical profession who are interested should apply for a special invitation as soon as possible.

Week-End Course at Wagga.

The Post-Graduate Committee, in conjunction with the Southern District Medical Association, will hold a week-end course at the Wagga Wagga Base Hospital on Saturday, April 17, and Sunday, April 18, 1948.

Saturday, April 17, 1948.

2 p.m.: Registration.
2.30 p.m.: "Fœtal and Neo-Natal Mortality", Dr. Mary Heseltine.
4 p.m.: "Psychiatric Problems in General Practice", Dr. John McGeorge.

Sunday, April 18, 1948.

10 a.m.: "Allergy of the Ear, Nose and Throat", Dr. Robert Steel.
11.30 a.m.: "Laboratory Investigations in Gynæcology", Dr. Mary Heseltine.

Wild flowers of Western Australia. (See "Congress Notes".)

2 p.m.: "Indications for Shock Therapy", Dr. John McGeorge.
3 p.m.: "Practical Aspects of Allergic Diseases", Dr. Robert Steel.

The fee for the course will be £1 1s. Those wishing to attend are requested to notify Dr. J. S. Storey, Honorary Secretary, Southern District Medical Association, Box S44, P.O., South Wagga, as soon as possible.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the Sixth Session of the Australasian Medical Congress (British Medical Association) to be held at Perth, Western Australia, from August 15 to 21, 1948, has forwarded the following information.

Office-Bearers of Sections.

The following is a supplementary list of office-bearers of sections, to be added to that published in this journal on October 4, 1947.

Section of Medicine: Vice-President, Dr. D. J. Thomas (Victoria).
Section of Naval, Military and Air Force Medicine and Surgery: Vice-President, Dr. H. G. Furnell (Victoria).
Section of Radiology and Radiotherapy: Vice-President, Dr. Keith Hallam (Victoria).
Section of Surgery: Vice-President, Dr. A. E. Lee (Queensland).

Plenary Session.

The programme for the plenary session, to be held on the morning of Tuesday, August 17, is now complete and is as follows:

Introduction: Dr. D. M. McWhae (President).

Papers: (*a*) "The Problem of the Control of Pulmonary Tuberculosis", Dr. H. W. Wunderly (Commonwealth) (20 minutes); (*b*) "The Treatment of Pulmonary Tuberculosis", Dr. L. I. Henzell (Western Australia) (20 minutes); (*c*) "Sociological Implications and Rehabilitation", Dr. D. R. W. Cowan (South Australia) (20 minutes); (*d*) "The Limitations of the Role of the Radiologist in the Diagnosis of Pulmonary Tuberculosis", Dr. Keith Hallam (Victoria) (20 minutes).

Discussion: (*a*) Dr. Alan King (Western Australia), "Control of Pulmonary Tuberculosis" (10 minutes); (*b*) Dr. W. Cotter Harvey (New South Wales), "Treatment of Pulmonary Tuberculosis" (10 minutes); (*c*) Dr. E. A. North (Victoria), "The Role of B.C.G. Vaccine in the Control of Pulmonary Tuberculosis" (10 minutes); (*d*) Dr. Milton Coutts (New South Wales), "Pulmonary Tuberculosis from the Point of View of the Bronchoscopist" (five minutes). All other speakers will be limited to five minutes.

Application for Membership.

Members of Branches who have not yet completed the form for membership of congress are asked to communicate with the Honorary Local Secretaries from whom forms may be obtained. The Honorary Local Secretaries are as follows:

New South Wales: Dr. K. B. Noad, c.o. the New South Wales Branch of the British Medical Association, 135, Macquarie Street, Sydney.
Queensland: Dr. Harold Love, Wickham House, Wickham Terrace, Brisbane.
South Australia: Dr. J. M. Bonnin, 175, North Terrace, Adelaide.
Tasmania: Dr. J. P. Millar, 163, Macquarie Street, Hobart.
Victoria: Dr. Roy F. Watson, Medical Society Hall, Albert Street, Melbourne.
New Zealand: The General Secretary, New Zealand Branch of the British Medical Association, Post Office Box 156, Wellington.

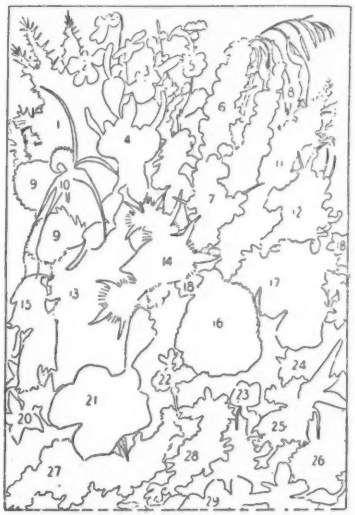
Accommodation has been asked for by a large number of practitioners who have not yet filled in their membership forms. The accommodation has been arranged, and the Executive Committee urges those intending members to forward their membership forms as soon as possible.

Accommodation.

The Executive Committee has heard of a number of practitioners who intend to visit Perth for the congress, but who have not asked for accommodation. While the Accommodation Committee is quite certain of accommodating everybody satisfactorily, its task is not an easy one. For this reason members who will attend congress are asked to make application for accommodation as soon as possible so as to avoid disappointment which may arise if a large number of applications come in at the eleventh hour.

Western Australian Wild Flowers.

As a special supplement to this issue there appears a coloured plate depicting some of the renowned Western Australian wild flowers. A line drawing of the illustration is included herewith and the key to the individual blossoms is as follows:



1. *Hypocalymma robusta* ..... Swan River Myrtle
2. *Lobelia tenuior* ..... Big Blue Lobelia
3. *Daviesia junceum* ..... Reed Butterflies
4. *Templetonia refusa* ..... Cockie's Tongues
5. *Eremophila eriocalyx* ..... Desert Pride
6. *Hovea trisperma* ..... Sand Hovea
7. *Hovea trisperma* ..... White Sand Hovea
8. *Kennedya nigricans* ..... Black Kennedya
9. *Pimelea suaveolens* .... Sweet-Scented Bunjong
10. *Caladenia Patersoni* ..... White Spider Orchid
11. *Hibbertia hypericoides* ..... Bush Primrose
12. *Grevillea Wilsoni* ..... Native Fuchsia
13. *Verticordia grandis* ..... Scarlet Featherflower
14. *Clematis pubescens* ..... White Clematis
15. *Blancoa canescens* ..... Winter Bell
16. *Banksia coccinio* ..... Scarlet Banksia
17. *Pimelea physodes* ..... Qualup Bell
18. *Platytheca galioides* ..... Blue Mist
19. *Wahlenbergia gracilis* ..... Bluebell
20. *Caladenia flava* ..... Cowslip Orchid
21. *Cienfugosia hakeæfolia* .. Red-Centred Hibiscus
22. *Oxylobium capitatum* ..... Bacon and Eggs
23. *Isotropis striata* ..... Granny Bonnet
24. *Conostephium pendulum* .... Pink-Tipped Pearl
25. *Marianthus ringens* ..... Orange Creeper
26. *Actinotus leucocephalus* ..... Flannel Flower
27. *Leschenaultia formosa* ..... Red Leschenaultia
28. *Pelargonium australe* ..... Wild Geranium
29. *Kennedya prostrata* ..... Red Runner

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1946*, of Queensland, as duly qualified medical practitioners:

de Vidas, Jack, M.R.A.C.P., 1942, 142, Lytton Road, East Brisbane.

Luke, Clifton James Herbert, M.B., 1947 (Univ. Sydney), Cairns Hospital, Cairns.

Suggit, Stephen Creighton, M.R.C.S. (England), L.R.C.P. (London), 1932, M.B., B.S., 1933 (Univ. London), F.R.C.S., 1934 (England), The Flat, 14, Florence Street, Ascot, Brisbane.

Suggit, Winifred Powys, M.R.C.S. (England), L.R.C.P. (London), 1931, M.B., B.S., 1933 (Univ. London), The Flat, 14, Florence Street, Ascot, Brisbane.

The following additional qualifications have been registered:

Livingstone, William Gordon, Glencairn, 149, Wickham Terrace, Brisbane, M.R.A.C.P., 1947.

McKillop, Lachlan Martin, Orient Line Buildings, Eagle Street, Brisbane, Ch.M. (Univ. Sydney), 1909, F.R.A.C.S., 1928, F.A.C.S., 1932.

The Royal Australasian College of Physicians.

LECTURES BY SIR HUGH CAIRNS.

AN invitation is extended to all members of the medical profession to attend two lectures by Sir Hugh Cairns, K.B.E., the first Commonwealth Travelling Professor under the Arthur Sims Foundation, to be given under the ægis of the Royal Australasian College of Physicians. Details of these lectures are as follows:

Friday, April 2, 1948, at 8.30 p.m. in the Stawell Hall of the Royal Australasian College of Physicians. Subject: "Treatment of Purulent Leptomeningitis."

Wednesday, April 7, 1948, at 8.30 p.m. in the Stawell Hall of the Royal Australasian College of Physicians. Subject: "Closed Head Injuries and their Effects."

Books Received.

"Stammering: Can It Be Cured", by H. C. Lendon Bell, with a foreword by Professor A. N. Burkitt, M.B., B.Sc.; 1948. Sydney: The Land Newspaper, Limited. 8¼" × 5½", pp. 36.

"Sickness Without Sorrow", by "G.P.", with illustrations by Alec Gurney; 1947. Melbourne: Robertson and Mullens, Limited. 8¼" × 5½", pp. 88, with many illustrations. Price: 6s.

"The 1947 Year Book of Urology", by Oswald S. Lowsley, M.D., F.A.C.S.; 1948. Chicago: The Year Book Publishers Incorporated. 7" × 4¾", pp. 414, with illustrations. Price: $3.75.

"Hormones and Behavior: A Survey of Interrelationships Between Endocrine Secretions and Patterns of Overt Response", by Frank A. Beach, with a foreword by Earl T. Engle; 1948. New York and London: Paul B. Hoeber Incorporated. 9¼" × 6¼", pp. 384. Price: $6.50.

"The 1947 Year Book of General Surgery", edited by Evarts A. Graham, A.B., M.D.; 1947. Chicago: The Year Book Publishers Incorporated. 7" × 4¾", pp. 734, with many illustrations. Price: $3.75.

"Diabetes Mellitus in General Practice", by Arthur R. Colwell, M.D.; 1947. Chicago: The Year Book Publishers Incorporated. 8" × 5½", pp. 350, with illustrations. Price: $5.25.

"The American Illustrated Medical Dictionary: A Complete Dictionary of the Terms Used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, Nursing, Veterinary Science, Biology, Medical Biography, etc., with the Pronunciation, Derivation and Definition", by W. A. Newman Dorland, A.M., M.D., F.A.C.S., with the collaboration of E. C. L. Miller, M.D.; Twenty-First Edition; 1947. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¼" × 6¼", pp. 1660, with many illustrations, some of them coloured. Price: 59s. 6d.

"Illustrative Electrocardiography", by Julius Burstein, A.B., M.D., and Nathan Bloom, M.D., F.A.C.P.; Third Edition; 1948. New York and London: D. Appleton-Century Company Incorporated. 9¼" × 6¼", pp. 326, with many illustrations.

"Aids to Gynæcology", by W. R. Witherton, M.A., M.B., B.Ch. (Camb.), F.R.C.S. (England), M.R.C.O.G; Tenth Edition; 1947. London: Baillière, Tindall and Cox. 6" × 4", pp. 190, with illustrations. Price: 5s.

"Medicine: Essentials for Practitioners and Students", by G. E. Beaumont, M.A., D.M. (Oxon.), F.R.C.P., D.P.H. (London); Fifth Edition; 1948. London: J. and A. Churchill, Limited. 9" × 6", pp. 848, with many illustrations, some of them coloured. Price: 30s.

"Studies on the Formation of Cellular Substances during Blood Cell Production", by B. Thorell; 1947. London: Henry Kimpton. 9" × 5¾", pp. 120, with many illustrations. Price: 12s.

Diary for the Month.

APRIL 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APRIL 7.—Western Australian Branch, B.M.A.: Council Meeting.
APRIL 7.—Victorian Branch, B.M.A.: Branch Meeting.
APRIL 9.—Queensland Branch, B.M.A.: Council Meeting.
APRIL 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APRIL 15.—Victorian Branch, B.M.A.: Organization Subcommittee.
APRIL 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
APRIL 20.—New South Wales Branch, B.M.A.: Medical Politics Meeting.
APRIL 21.—Western Australian Branch, B.M.A.: General Meeting.
APRIL 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
APRIL 22.—Victorian Branch, B.M.A.: Executive Meeting.
APRIL 23.—Queensland Branch, B.M.A.: Council Meeting.
APRIL 27.—New South Wales Branch, B.M.A.: Ethics Committee.
APRIL 28.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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